Animal Production Science, 2013, **53**, 181–208
http://dx.doi.org/10.1071/AN12048

Review

A review of potential contaminants in Australian livestock feeds and proposed guidance levels for feed

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Abstract. Contaminants of man-made and natural origin need to be managed in livestock feeds to protect the health of livestock and that of human consumers of livestock products. This requires access to information on the transfer from feed to food to inform risk profiles and assessments, and to guide management interventions such as regulation or Hazard Analysis Critical Control Point approaches. This paper reviews contaminants of known and potential concern in the production of livestock feeds in Australia and compares existing but differing state and national regulatory standards with international standards. The contaminants considered include man-made organic chemical contaminants (e.g. legacy pesticides), elemental contaminants (e.g. arsenic, cadmium, lead), phytotoxins (e.g. gossypol) and mycotoxins (e.g. aflatoxins). Reference is made to scientific literature and evaluations by regulators to propose maximum levels that can be used for guidance by those involved in managing contamination incidents or developing feed safety programs.

Received 10 February 2012, accepted 6 September 2012, published online 8 January 2013

Introduction

Contaminants in feed for livestock need to be managed to protect livestock health, and to minimise residues in livestock products that might affect the health of human consumers or impair marketing and international trade. Contaminants can arise from man-made organic chemical sources such as pesticides, from environmental sources of contamination (Gilbert and Şenyuva 2005), or from natural toxins present in plants (phytotoxins) and fungi (mycotoxins) (Blaney 2005). Feed contamination has caused major food safety incidents in Europe, such as dioxin and polychlorinated biphenyl (PCB) contamination of poultry products in Belgium and lead in feed of dairy cattle in the Netherlands (Baars *et al*. 1992; Covaci *et al*. 2008). On several occasions Australian livestock producers and government authorities have also had to deal with feed and environmental contamination affecting trade, but these incidents have generally been well managed and no significant risks to human health have resulted. To maintain this record requires that systems for control and management of feed safety should be periodically re-evaluated and strengthened where necessary.

Regulation by government has been for many years a key process for managing contaminants and is likely to remain so where there are perceived risks to human health. Where the risks are primarily to livestock production and to trade, government policy is trending towards industry self-regulation. Feed safety systems should aim to ensure that food derived from animals is suitable for human consumption. This is achieved by adherence to good animal feeding practice at the farm level, and good manufacturing practices during procurement, handling, storage, processing and distribution of animal feed, feed additives and feed ingredients. One suitable process for such management is that of Hazard Analysis Critical Control Point, a systematic approach to the identification, assessment and control of hazards in the food chain. However, in many instances the information necessary to assess the risk of various contaminants is not readily accessible, hampering attempts to use pre-emptive risk management strategies.

The aim of the present paper is to review knowledge of contaminants in Australia, and to then recommend guidance maximum levels for livestock feed used for food-producing species. This will improve compliance with human food standards and minimise risks to consumers' health, to livestock health and productivity, to trade in animal products, and to trade in animal feed. The scope of the paper is limited to cattle, sheep, goats, pigs and poultry, the main livestock species used in Australia to produce food.

The main classes of contaminants of feed considered here are:

(1) Man-made organic chemical contaminants (e.g. dieldrin, dioxins);
(2) Elemental contaminants (e.g. arsenic, cadmium, lead, mercury);

(3) Phytotoxins (e.g. gossypol); and
(4) Mycotoxins (e.g. aflatoxin B1)

Guidance levels for contaminants should be set at concentrations in animal feeds such that their use will not result in violations of the current regulatory standards for food of animal origin (edible tissues, milk or eggs). A recent literature review has summarised factors for the transfer of contaminants from feed to tissues, milk or eggs (MacLachlan 2011). The reported transfer factors (TFs), sometimes also known as concentration ratios, can be used to estimate guidance levels for feed. They are based on long-term exposure of livestock to a contaminant such that the levels in tissues, milk or eggs approach a constant value with time.

$$TF_i = C_i/C_{feed} \quad (1)$$

where C_i is the contaminant concentration in the animal commodity of interest (muscle, fat, liver, kidney, milk, eggs) with units mg/kg fresh weight and C_{feed} is the contaminant concentration in the animal diet (mg/kg DM).

The TF can be used to calculate the concentration of a contaminant in feed that will lead to a particular level in tissues, milk or eggs.

$$C_{feed} = C_i/(R \times TF_i) \text{ where i} = \text{fat, muscle, liver, kidney, milk or eggs} \quad (2)$$

where R = bioavailability of the source relative to material used in transfer studies and bioavailability is the fraction ingested that is absorbed (if unknown it is assumed R = 1).

In recommending guidance levels for livestock feeds, consideration should be given to existing industry-based standards and government regulations. Where possible, the proposed guidance levels should harmonise existing regulations and industry standards.

Man-made organic chemical contaminants

A range of man-made organic chemicals can be found in livestock feeds such as pesticides applied to crops that might consequently be used as feed, the carry-over of veterinary medicine residues from medicated to non-medicated feed and the contamination of feed with chemicals found in the environment. Compounds that have both natural and man-made sources are also included in this section (dioxins, polycyclic aromatic hydrocarbons, PAH). The health of livestock is usually unaffected at the concentrations found in feeds; rather it is the transfer to food of animal origin (tissues, milk and eggs) that is of concern.

Agricultural pesticides and veterinary medicines are assessed as part of the regulated registration process by the Australian Pesticides and Veterinary Medicines Authority (APVMA). The registration process includes consideration of the potential for residues in livestock arising from residues in feed. The livestock industry has conducted additional assessments for these compounds based on either meeting or reducing the risk of not complying with differing standards in export markets. Consequently, it is considered that current pesticides and veterinary medicines are adequately addressed within the current system (MacLachlan and Bhula 2008; Lutze *et al*. 2009).

Because of their intake of pasture and roughages as well as possible ingestion of contaminated soil ruminants (cattle, sheep and goats) are vulnerable to background environmental contamination. In contrast, pigs and poultry (as well as lot-fed cattle and sheep) are fed controlled diets and are more susceptible to contaminated feed ingredients such as feed additives used in manufactured feed.

The continued legacy of organochlorine pesticides in the environment resulting from legal usage many years ago in Australia, as well as their ongoing use in some countries, can cause exposure through feed and result in their accumulation in fatty tissues, milk and eggs of livestock. The main source of exposure in Australia is through access by livestock to historically contaminated land rather than traded feed (Robertson *et al*. 1990). Cattle are the main species affected and residues in beef cost the industry hundreds of millions of dollars in testing costs and lost trade in the 1980s and 1990s (Shaw and Eustace 1993). Since then residues are adequately controlled through the National Organochlorine Residue Management program and on-farm risk assessments conducted as part of the industry on-farm food safety and quality assurance programs (LPA 2011). Additional exposure through manufactured feeds should be controlled to ensure residues are minimised.

Table 1 lists maximum limits (MLs) for food of animal origin for various man-made organic chemical contaminants and includes levels for Australia and major markets for Australian livestock commodities. As 60–70% of Australian production of beef and sheep meat is exported, it is important that guidance levels for feed minimise the risk of exceeding standards in major markets.

Organochlorine pesticides

Maximum levels for persistent organochlorine pesticides (DDT, dieldrin, hexachlorobenezene, heptachlor and lindane) in feed have been established by individual states and territories within Australia (Vic. 1992; Qld 1997; WA 2006; NSW 2010) and are consistent with the production of food that will comply with relevant MLs in the Australia New Zealand Food Standards Code (the Food Code). Similar values for stock feed are listed in the APVMA MRL Standard. International maximum residue limits (MRLs) for persistent organochlorine pesticides in food are similar to the Australian values. Monitoring conducted by the National Residue Survey (NRS) indicates that detectable residues in species other than cattle are infrequent (<0.1%), and non-compliant residues are rare in all species.

The current standards in Australia for livestock feed in the APVMA MRL Standard are suitable with the exception of mirex for which no standard has been established. A TF for mirex in fat of 1.9 has recently been reported (MacLachlan 2011). Relative bioavailability is assumed to be one. If encountered in feed, making use of Eqn 2 it is suggested that levels of mirex in the total diet be less than 0.01 mg/kg ÷ 1.9 = 0.005 mg/kg DM to ensure the concentration in fat of exposed animals is less than 0.01 mg/kg, a default concentration applied in several markets including the EU and Japan.

Table 1. Maximum limits and action levels (mg/kg wet weight except dioxins, which are on a ng TEQ[A]/kg fat basis and indicator PCBs, which are on a μg/kg fat basis) for man-made organic chemical contaminants in various countries and regions

c = cattle, p = pigs, po = poultry, ch = chicken, b = beef, s = sheep, fat = residue should be measured in trimable fat in the case of meat and in mechanically separated milk fat in the case of whole milk, F = the residue is fat soluble and when measured on a milk fat basis would be up to 25× higher

Contaminant	Liver	Kidney	Meat	Milk	Eggs	Country/region	Reference
DDT[E]	5	5	5 fat	1.25 fat	0.5	Australia	FSANZ (2012)
DDT[F]	–	–	2 fat	0.02	0.1	China	China (2005*a*)
DDT[E]	–	–	5 fat	0.02 F	0.1	Codex	Codex (2011)
DDT[E]	1	1	1 fat	0.04	0.05	EU	EU (2011*a*)
DDT[E]	2	2	5 (2 ch) fat	0.02	0.1	Japan	Japan (2011*a*)
DDT[E]	–	–	2 fat	0.02 F	0.1	Korea	Korea (2009)
DDT[G]	–	–	0.1	0.05	0.1	Russia	Russia (2010)
DDT[E]	–	–	5 fat	0.006 F	0.1	Taiwan	Taiwan (2009*a*)
DDT[H]	–	–	–	1.25 fat	0.5	USA	USFDA (2011)
Dieldrin[I]	0.2	0.2	0.2 fat	0.15 fat	0.1	Australia	FSANZ (2012)
Dieldrin[I]	–	–	0.2 fat	0.006 F	0.1	Codex	Codex (2011)
Dieldrin[I]	0.2	0.2	0.2 fat	0.006	0.02	EU	EU (2011*a*)
Dieldrin[I]	0.2	0.2	0.2 fat	0.006	0.1	Japan	Japan (2011*a*)
Dieldrin[I]	–	–	0.2 fat	0.006 F	0.1	Korea	Korea (2009)
Dieldrin[I]	–	–	0.2 fat	0.006 F	0.1	Taiwan	Taiwan (2009*a*)
Dieldrin[I]	–	–	–	0.3 fat	–	USA	USFDA (2011)
HCB	1	1	1 fat	0.5 fat	1	Australia	FSANZ (2012)
HCB	0.2	0.2	0.2 fat	0.01	0.02	EU	EU (2011*a*)
HCB	0.6	0.6	0.6 fat	0.01	0.5	Japan	Japan (2011*a*)
Heptachlor[J]	0.2	0.2	0.2 fat	0.15 fat	0.05	Australia	FSANZ (2012)
Heptachlor[J]	–	–	0.2 fat	0.006 F	0.05	Codex	Codex (2011)
Heptachlor[J]	0.2	0.2	0.2 fat	0.004	0.02	EU	EU (2011*a*)
Heptachlor[J]	0.2	0.2	0.2 fat	0.006	0.05	Japan	Japan (2011*a*)
Heptachlor[J]	–	–	0.2 fat	0.006 F	0.05	Korea	Korea (2009)
Heptachlor[J]	–	–	0.2 fat	0.006 F	0.05	Taiwan	Taiwan (2009*a*)
Heptachlor[J]	–	–	–	–	0.05	USA	USFDA (2011)
Lindane	2	2	2 fat	0.2 fat	0.1	Australia	FSANZ (2012)
Lindane	–	–	1 fat	0.01	0.1	China	China (2005*a*)
Lindane	0.01	0.01	0.1 fat	0.01	0.01	Codex	Codex (2011)
Lindane	0.02	0.02	0.02 fat	0.001	0.01	EU	EU (2011*a*)
Lindane	1 c 0.01[B]	1 c 0.01[B]	3 c 0.1[B]	0.01	0.01	Japan	Japan (2011*a*)
Lindane	–	–	2 fat	0.01 F	0.1	Taiwan	Taiwan (2009*a*)
Lindane	–	–	–	0.3 fat	0.5	USA	USFDA (2011)
Dioxins[K]	4.5 (10)	–	2.5 (4[C]) b s 1.75 (3[C]) po 1 (1.25[C]) p	2.5 (5.5)	2.5 (5)	EU	EU (2011*b*)
Dioxins[L]	–	–	4 b 2 p 3 ch	–	–	Korea	Korea (2011)
Dioxins[L]	6	6	3	3	3	Russia	Russia (2010)
Polychlorinated biphenyl (PCB) (total)	–	–	0.2 fat	0.2	0.2	Australia	FSANZ (2012)
PCB (indicator)	40[M]	–	40[M]	40[M]	40[M]	–	EU (2011*b*)
PCB (total)	–	–	0.5	0.1	0.2	Japan	Japan (2011*b*)
PCB (total)	–	–	1 fat	0.5 fat	0.2	Taiwan	Taiwan (2009*b*)
PCBs (total)	–	–	3 fat	–	–	USA	USFDA (2011)
Benzene(a)pyrene	–	–	–	1[D]	–	EU	EC (2006*b*)
Melamine	2.5	2.5	2.5	1	2.5	Codex	Codex (2010)

[A]Dioxins and dioxin-like (dl)-PCB are regulated as a group using the concept of toxic equivalents (TEQ). To obtain the residue expressed in terms of TEQ, the concentrations for individual congeners are multiplied by the congener toxic equivalency factor (TEF) and summed, i.e. $\Sigma(C_i \times TEF_i)$.

[B]All other species. [C]Figures in brackets are limits including dl-PCB. [D]Infant milk. [E]Sum of p,p′-DDT + o,p′-DDT+ p,p′-DDE + p,p′-DDD.

[F]Sum of p,p'-DDT + p,p'-DDE, + p,p'-DDD. [G]Sum of DDT and its metabolites. [H]Sum of DDT, DDE and DDD. [I]Sum of HHDN + HOED.

[J]Sum of heptachlor + heptachlor epoxide expressed as heptachlor.

[K]WHO TEF for human risk assessment based on the conclusions of the World Health Organisation meeting in Geneva, Switzerland in June 2005 as listed in van den Berg *et al*. (2006).

[L]WHO TEF for human risk assessment based on the conclusions of the World Health Organisation meeting in Stockholm, Sweden, 15–18 June 1997 as listed in van den Berg *et al*. (1998).

[M]Sum of PCB28 + PCB52 + PCB101 + PCB138 + PCB153 + PCB180.

Dioxin and dioxin-like polychlorinated biphenyls

Contamination of feed and feed ingredients with dioxins and dioxin-like PCBs (dl-PCBs) has caused billions of dollars of damage to livestock producers in Europe, the USA and Chile in the last 30 years (Buzby and Chandran 2003; Kim *et al*. 2009; Heres *et al*. 2010; Alcoser *et al*. 2011). Dioxins can be present in feed due to both natural and man-made sources (Fries 1995). Sources of contamination have included ball clay used as an anticaking agent in feed, lime as a neutralisation agent for citrus pulp, waste oil, choline chloride, zinc and copper oxides, gelatine, recycled oil and contaminated oils containing PCB used to heat air for drying bread crumbs and maize, and most recently contaminated fatty acids. In none of these cases was the contamination foreseen.

The public health and safety risk for Australians from exposure to dioxins from foods is very low and there are no Australian standards for dioxins and dl-PCBs in foods of animal origin or in animal feeds (FSANZ 2004). Several major markets for Australian exports have established MLs for animal commodities with many of the standards similar to those of the EU (EU 2011*b*). While the risk of dioxins in Australian livestock is considered low (DAFF 2004) it is advisable that feed ingredients be monitored against the MLs present in the EU to ensure levels of dioxins are as-low-as-reasonably-achievable (EC 2006*a*). It should be noted that the transfer of dioxin residues into food of animal origin depends on the congener profile of the feed. TF for typical environmental sources of dioxins present in feed for cattle, pig and chicken fat are estimated to be 5.3, 3.7 and 8.8, respectively, (MacLachlan 2011) suggesting levels in the total diet should be kept below 0.5 ng toxic equivalents (TEQ)/kg for cattle, 0.3 ng TEQ/kg for pigs and 0.2 ng TEQ/kg for poultry. These values are lower than the EU ML of 1.5 ng TEQ/kg for the total diet. The maximum level of dioxins that can be accommodated in the diet and for which derived food of animal origin will comply with EU standards will depend on the congener profile of the dioxin residue as well as bioavailability.

Polychlorinated biphenyls

Following exposure of cattle to feed containing PCBs in general, concentrations in fat can be 4.3 times greater than in feed (MacLachlan 2011) suggesting PCBs should not be present in the total diet at levels greater than 0.05 mg/kg (0.2 mg/kg ÷ 4.3 = 0.047 mg/kg).

Polybrominated biphenyls

The standard in New South Wales (NSW 2010) and Western Australia (WA 2006) for polybrominated biphenyls (PBBs) is nil for manufactured stock foods. No standards have been established in the Food Code for PBBs in food. European Food Safety Authority (EFSA) concluded that health-based standards were not required for PBBs (EFSA 2010*a*). At this stage it does not seem justified to recommend a guidance level for PBB given their ubiquitous presence in the environment and the lack of a human health concern.

Other man-made organic compounds

A few substances are of potential concern because of their widespread usage in human communities and ability to produce residues in food of unknown health risk. These include acrylamide, chlorinated paraffins, melamine, mineral hydrocarbons, perfluorinated alkanes, phthalates, polybrominated flame retardants and PAHs. Of these, standards for food of animal origin have only been established internationally for PAHs and melamine.

Benzo[a]pyrene has been used in the EU as a marker for PAH residues in food (EC 2006*b*). The transfer of benzo[a]pyrene to tissues, milk and eggs is negligible and as such guidance levels are not required for feed. However, hydroxylated metabolites of PAHs are found at significant concentrations in tissues, milk and eggs of exposed animals and if regulatory concern shifts to include hydroxylated metabolites guidance levels may be required.

The Codex Committee on Contaminants in Food has recommended an ML for melamine in animal feed of 2.5 mg/kg, noting that the ML applies to levels of melamine resulting from its non-intentional and unavoidable presence in feed (Codex 2010). The ML does not apply to feed for which it can be proven that concentrations of melamine higher than 2.5 mg/kg are the consequence of either the authorised use of cyromazine as an insecticide, or derived from the legitimate use of the feed ingredients guanidino acetic acid, urea and biuret. Transfer studies with lactating dairy cows indicate concentrations in milk and tissues are not expected to exceed maximum levels set by the Codex Alimentarius Commission (Codex) when feed contains concentrations of up to 30 mg/kg feed, a substantial margin of safety. The limit established by Codex for feed is based on residues being as-low-as-reasonably-achievable. The maximum level is also below that which might result in an adverse effect on livestock production as noted in a recent review of the literature by the EFSA (2010*b*). This review concluded that exposures to melamine and related compounds individually at scenarios of up to 10 mg/kg in feed are well below the doses causing toxicity and are therefore not expected to pose a risk to livestock.

State and APVMA MLs and proposed guidance maximum levels for legacy pesticides and environmental contaminants are summarised in Table 2.

Elemental contaminants

Food safety authorities have established MLs for a small number of elements in a variety of foods, including animal commodities when these are major contributors to human exposure. Human health risks arise from the persistence and accumulation of these elements in human tissues until a toxic threshold is reached. The major elements of concern for livestock health and/or residues in meat are cadmium, lead, mercury, arsenic, fluorine and potentially chromium, nickel, copper, zinc, selenium and molybdenum. The 20th Australian Total Diet Survey (FSANZ 2003) investigated the dietary exposure of the Australian population to different elements: antimony, arsenic, cadmium, lead, mercury, copper, selenium, zinc and tin, the 22nd Australian Total Diet Survey (FSANZ 2008) investigated chromium, molybdenum, nickel and selenium while the 23rd Australian

Table 2. Current state or Australian Pesticide and Veterinary Medicines Authority (APVMA) maximum limits (MLs) and recommended guidance maximum levels for man-made organic chemical contaminants in feed
n = New South Wales; q = Queensland; v = Victoria; w = Western Australia

Contaminant	Feed	State or APVMA ML (mg/kg DM)	Recommended guidance maximum level (mg/kg DM)
Aldrin (Σ HHDN + HEOD)	Total diet	0.01 nqvw	0.01
BHC (other than γ-BHC)	Total diet	0.02 nqvw	0.02
Chlordane	Total diet	0.01 nqvw	0.01
DDT[A]	Total diet	0.05 nqvw	0.05
Dieldrin (Σ HHDN + HEOD)	Total diet	0.01 nqvw	0.01
Endrin	Total diet	0.03 nqvw	0.03
HCB	Total diet	0.01 nqvw	0.01
Heptachlor (Σ heptachlor + epoxide)	Total diet	0.02 nqvw	0.02
Lindane (= γ-BHC)	Total diet	0.1 nqvw	0.1
Mirex	Total diet	–	0.005
Sum of the adulterants listed	Total diet	0.1 nqw	0.1
PBB (Σ congeners)	Total diet	0 nw	–
Polychlorinated biphenyl (PCB) (Σ congeners)	Total diet	0.05 nw	0.05
Dioxins + dl-PCBs (Σ TEQ)[B]	Total diet for cattle, sheep, goats	–	0.5 ng TEQ/kg DM
	Total diet for pigs	–	0.3 ng TEQ/kg DM
	Total diet for poultry	–	0.2 ng TEQ/kg DM
	Feed ingredients except as below:	–	1.25 ng TEQ/kg DM
	Vegetable oils, minerals, trace elements, premixes, anti-caking agents and binders	–	1.5 ng TEQ/kg DM
	Animal fats, milk fats, egg fats	–	3 ng TEQ/kg DM
	Fish, other aquatic animals and their products except as below:	–	4.5 ng TEQ/kg DM
	Fish oil	–	24 ng TEQ/kg DM
	Fish protein hydrolysates with >20% fat	–	11 ng TEQ/kg DM
Melamine	Total diet	–	2.5

[A]DDT = Σ(p,p′-DDT + o,p′-DDT+ p,p′-DDE + p,p′-DDD).
[B]Dioxins and dioxin-like-PCBs are regulated as a group using the concept of toxic equivalents (TEQ). To obtain the residue expressed in terms of TEQ, the concentrations for individual congeners are multiplied by the congener toxic equivalency factor (TEF) and summed, i.e. $\Sigma(C_i \times TEF_i)$ (van den Berg *et al*. 2006).

Total Diet Survey (FSANZ 2011) included arsenic, chromium, cadmium, copper, fluoride, lead, mercury, selenium, and zinc. In all cases FSANZ concluded there was no evidence to suggest that intakes of these elements by the Australian population exceeded safe levels.

Like all potentially toxic elements, these elements are also tolerated only up to a certain limit by animals. Above that limit, their intrinsic toxic potential leads to detrimental effects. Exposure to, and requirement for, elements are influenced by factors such as the class of animal, level of production, chemical form of the element, mineral interrelationships, dietary intake, breed differences and adaptation on long-term exposure. The National Research Council (NRC) of the USA has established maximum tolerable levels (MTLs) for different minerals in the diets of livestock. These MTLs are the 'dietary level that, when fed for a defined period of time, will not impair animal health or performance' (NRC 2005).

Table 3 lists MLs for food of animal origin for various chemical elements and includes levels for Australia and major markets for Australian livestock commodities together with Generally Expected Levels (GELs) (90th percentiles). The latter have been proposed by FSANZ to assist food manufacturers in ensuring concentrations of these contaminants are as-low-as-reasonably-achievable (FSANZ 2001). Concentrations above the GEL should prompt an investigation to determine whether or not samples contain consistently high concentrations and to investigate whether management changes can be implemented to reduce these. Concentrations above the GEL do not indicate the food is unsafe.

The following discussion concentrates only on ensuring compliance with established MLs and GELs (90th percentiles). The use of some chemical elements such as copper and chromium for growth promotion, or zinc as a medication, is not considered. In calculation of guidance maximum levels for feed, TFs were taken from the recent review by MacLachlan (2011). When recommending maximum levels for the total diet of livestock the bioavailability of elements in feed relative to the forms used in studies on the transfer to tissues, milk and eggs was assumed to be 1.0.

Antimony

Concentrations of antimony in the environment are low with major sources associated with mining and application of biosolids to agricultural land (NRC 2005). Only low concentrations of antimony have been found in monitoring studies of meat (NRS 1997). TFs of 0.18 have been reported for cattle kidney and muscle (MacLachlan 2011). FSANZ have recommended GELs

Table 3. Maximum limits and 90th percentile Generally Expected Levels (GELs, mg/kg wet weight) for various elements in livestock commodities in various countries or regions[A]

s = sheep, ch = chicken

Element	Liver	Kidney	Meat	Milk	Eggs	Country/region	Reference
Antimony	0.05	0.05	0.05	–	–	Australia (GEL)	FSANZ (2001)
Arsenic[B]	0.1 (1 ch)	0.1	0.02	–	–	Australia (GEL)	FSANZ (2001)
Arsenic[B]	–	–	0.5	0.1	–	China	China (2005*b*)
Arsenic[B]			0.1 fat	–	–	Codex	Codex (2010)
Arsenic[B]	1	1	0.1	0.05	0.1	Russia	Russia (2010)
Cadmium	1.25	2.5	0.05	–	–	Australia	FSANZ (2012)
Cadmium	0.5	1	0.1	–	0.05	China	China (2005*b*)
Cadmium	0.5	1	0.05	–	–	EU	EC (2006*b*)
Cadmium	0.3	1	0.05	0.03	0.01	Russia	Russia (2010)
Chromium	1	1	1	0.3	1	China	China (2005*b*)
Copper	50 (150 s)	50	2	–	–	Australia (GEL)	FSANZ (2001)
Lead	0.5	0.5	0.1	–	–	Australia	FSANZ (2012)
Lead	0.5	0.5	0.2	0.05	0.2	China	China (2005*b*)
Lead	0.5	0.5	0.1	0.02	–	Codex	Codex (2010)
Lead	0.5	0.5	0.5	0.02	–	EU	EC (2006*b*)
Lead	0.6	1.0	0.5	0.1	0.3	Russia	Russia (2010)
Lead	0.5	0.5	–	–	–	Taiwan	Taiwan (2009*c*)
Mercury	0.01	0.01	0.01	–	–	Australia (GEL)	FSANZ (2001)
Mercury	–	–	0.05	0.01	0.05	China	China (2005*b*)
Mercury	0.1	0.2	0.03	0.005	0.02	Russia	Russia (2010)
Selenium	2	2	0.2	–	–	Australia (GEL)	FSANZ (2001)
Zinc	60	60	75	–	–	Australia (GEL)	FSANZ (2001)

[A]Japan, the Republic of Korea and the USA have not established limits for the metals listed in livestock commodities.
[B]Total arsenic.

of 0.05 mg/kg for meat of cattle, pigs and sheep and 0.05 mg/kg for edible offal of cattle, pigs and sheep (Table 3). To have confidence that concentrations in tissues will be below the 90th percentile GEL in Australia, it is recommended that concentrations in feed should not exceed 0.05 mg/kg ÷ 0.18 = 0.28 mg/kg DM. On rounding the guidance maximum level is 0.3 mg/kg DM for antimony in the total diet of cattle, pigs and sheep.

Arsenic

Sources of arsenic for grazing livestock include contaminated land associated with its historic use as an acaricide for tick control, and some natural outcrops of arsenical mineral deposits. However, only low concentrations of arsenic in livestock products have been found in monitoring studies (NRS 1997). The transfer of arsenic from feed to tissues, milk and eggs depends on the arsenic compound administered, animal species and duration of exposure. TFs of 0.091 and 0.13 for total arsenic have been reported for cattle muscle and cattle milk, respectively (MacLachlan 2011). FSANZ have recommended 90th percentile GELs of 0.02 mg/kg for meat of cattle, pigs and sheep, 0.1 mg/kg for edible offal of cattle, pigs and sheep and 1 mg/kg for liver of chickens (Table 3). To have confidence that concentrations in muscle are below the GEL in Australia, concentrations in feed should not exceed 0.02 mg/kg ÷ 0.091 = 0.2 mg/kg DM. For lactating animals, concentrations of arsenic in feed should be below 0.05 mg/kg ÷ 0.13 = 0.4 mg/kg DM to be confident concentrations in milk will be below the standard in Russia. Since the estimated level for meat animals is lower than that required for lactating animals, the maximum level proposed for cattle, pigs and sheep feeds is 0.2 mg/kg DM.

Cadmium

Cadmium is a ubiquitous contaminant that is present in many feed and feed ingredients (EFSA 2004*a*). The main source of livestock exposure for animals produced under extensive systems is through the environment (forage and soil) while for production under intensive systems the major sources are feed ingredients (EFSA 2004*a*).

Grain-based feed materials generally contain low concentrations of cadmium. Data on the cadmium content of several cereal grain, pulse and oilseed feed materials have been reported (NRS, pers. comm.). In most (>99.9%) samples cadmium was not detected above the limit of reporting of 0.01 mg/kg. Cadmium impurities can be present in mineral-based feed materials such as phosphates including phosphatic fertilisers, and can be a significant contributor to livestock dietary intake (EFSA 2004*a*).

Cadmium disposition is significantly influenced by dietary interactions with zinc, copper, iron and calcium (Suttle 2010). Ascorbic acid and cholecalciferol can influence the rate of absorption. Additionally, the absorption of dietary cadmium depends on the cadmium concentration in individual feed materials, time and frequency of exposure, the animal species, animal age or stage of development, and nutritional status of the animal (Suttle 2010).

As environmental cadmium exposure for grazing animals can be substantial, additional exposure through manufactured feeds

should be controlled and kept to a minimum to reduce the chance that tissue concentrations exceed MLs in tissues at slaughter. Little data is available on cadmium concentrations in feed additives used in Australia, however many feed additives are traded internationally and information is available from a recent EFSA review (EFSA 2004*a*). Cadmium contents of mineral supplements and premixtures ranged from <0.01 to 2.3 mg/kg DM with a mean value of 0.6 mg/kg DM.

Advice on maximum levels for exposure to cadmium are challenging to develop as cadmium accumulates with increasing duration of exposure, and the slaughter of livestock usually occurs before steady-state concentrations are achieved in tissues. In this case the use of a TF is not ideal. Additionally the Australian ML for cadmium for kidney and liver listed in the Food Code (FSANZ 2012) are higher than those for some significant markets.

Concentrations of cadmium in the total diet should be as-low-as-reasonably-achievable and for ruminants not greater than 1 mg/kg DM. Based on the results of the EFSA review, levels are also proposed as listed in Table 4 for cadmium in various feed ingredients.

Australia has developed a strategy to reduce the concentrations of cadmium found in meat and meat products which should assist producers in keeping cadmium tissue concentrations as-low-as-reasonably-achievable (Safemeat 2007).

Chromium

While naturally present in the environment at low concentrations, phosphate added to rations is thought to be the major source of chromium in livestock diets (NRC 2005; Suttle 2010). Chromium is also sometimes added to diets as a supplement (Suttle 2010). The absorption of Cr(III) is poor and tissue residues are generally very low (NRC 2005; Suttle 2010). There are no specific regulations on maximum levels of chromium in foods in Australia. China has established standards at 1 mg/kg wet weight (WW) for liver, kidney, muscle and eggs and 0.3 mg/kg for milk. Using a TF for cattle liver of 0.055 (MacLachlan 2011), it is recommended that the maximum level at which chromium should be incorporated into the total diet for compliance with the standard in China should be 18 mg/kg DM (1 mg/kg ÷ 0.055 = 18 mg/kg), which can be rounded to 20 mg/kg DM.

Copper

Apart from deliberate feed supplementation, sources of copper for livestock include mineral deposits, fertilisers, and its use to control fungi in crops, preserve wood, and control cyanobacteria in water supplies (NRC 2005; Suttle 2010). Copper absorption and utilisation by livestock can be markedly affected by several mineral elements and other dietary factors. Zinc, iron, molybdenum, inorganic sulfate and other nutrients can reduce copper absorption (Suttle 2010). FSANZ has established GELs of 50 mg/kg WW for edible offal of cattle and pigs and 150 mg/kg WW for sheep liver that can be used to derive guidance levels for feed (Table 3). A TF of 2.4 has been reported for cattle liver (MacLachlan 2011) suggesting concentrations in cattle feed should not exceed 50 ÷ 2.4 = 21 mg/kg DM. A level of 20 mg/kg DM should be adequate to ensure copper concentrations do not exceed 50 mg/kg WW in cattle liver. Based on a GEL for sheep liver of 150 mg/kg WW and a TF of 5 (MacLachlan 2011), concentrations in the total diet for sheep should not exceed 150 ÷ 5 = 30 mg/kg DM. The calculated maximum level for copper in the total diet of sheep is higher than the MTL recommended by the NRC (2005) of 15 mg/kg DM. As the MTL is lower than the calculated level, the level of copper in the total diet of sheep should not exceed the MTL of 15 mg/kg DM.

Pigs do not store as much copper in liver as ruminants (Suttle 2010). Based on a GEL of 50 mg/kg WW for edible offal of cattle and pigs and a TF of 0.58 for pig liver (MacLachlan 2011), concentrations in the total diet should not exceed 50 ÷ 0.58 = 86 mg/kg DM while the calculation for pig muscle suggests copper concentrations in feed should not exceed 133 mg/kg. A level of 100 mg/kg DM for the total diet should be adequate to ensure copper in pig tissues do not significantly exceed GEL (90th percentile) levels.

Copper has been incorporated in rations of pigs and poultry at concentrations of up to 50–250 mg/kg DM for growth promotion, though in the case of pigs the concentration is usually decreased to 5 mg/kg DM after the pigs reach 55 kg liveweight (Jacela *et al*. 2010). It is noted that the levels in current state regulations take into account use of copper for growth promotion and as such are higher than the guidance maximum levels recommended here.

Fluoride

Neither MLs nor GELs have been established for fluoride in livestock commodities. It has been reported that the major source of fluorides for grazing livestock in Australia is artesian water supplies, which can contain 1–10 mg fluoride/L. Artesian water has been associated with endemic fluorosis in sheep in localised areas of Queensland (Harvey 1952). The other major source of fluoride is rock phosphates and fertilisers derived from rock phosphates, which are commonly incorporated into stock feeds as phosphate sources. It is to prevent fluorosis in stock from the latter sources, that some states have regulations specifying maximum levels of fluoride in stock feed, which must be observed in these jurisdictions. The limits for fluoride in Queensland (Qld 1997) and Western Australia (WA 2006) are similar. The limits in Queensland for the total diet are 40 mg/kg for dairy cattle, 150 mg/kg for breeding pigs, 350 mg/kg for poultry and 200 mg/kg for other livestock. Fluoride accumulates in teeth and bone, and concentrations of fluoride in edible tissues are very low (Puls 1994). It is not proposed to recommend levels different to those currently in state regulations.

Lead

Lead has been a common cause of cattle poisoning in Australia over the last century through use of lead-based paint on stock yards and buildings and through consumption of (apparently sweet-tasting) lead salts derived from discarded sump oil and lead/acid batteries, but these sources are now much better controlled (Burren *et al*. 2010; Byrne and Gill 2011). Lead impurities are often present in mineral feed materials such as phosphates and oxides, and can contribute significantly to livestock dietary exposure (NRC 2005). Lead is a chronic and cumulative poison and human exposure should be as-low-as-reasonably-achievable (NRC 2005). Lead is absorbed to a different extent depending on various factors (intake,

Table 4. Current state and recommended guidance maximum levels for various elements in feed
n = New South Wales; q = Queensland; w = Western Australia

Element, species	Feed	State maximum limits (mg/kg DM)	Recommended guidance maximum level (mg/kg DM)
Antimony			
Cattle, pigs, sheep	Total diet	–	0.3
Arsenic (total)			
Cattle, sheep, pigs	Total diet	–	0.2
Cadmium			
Cattle, poultry, sheep	Total diet	–	1
Pigs	Total diet	0.5 nqw	0.5
All	Ingredients of vegetable origin	–	1
All	Ingredients of animal origin	–	2
All	Feed additives of mineral origin – phosphates	100[A] qw	20
All	Feed additives of mineral origin – other than phosphates		2
All	Trace element additives based on copper oxide, manganous oxide, zinc oxide or manganous sulfate monohydrates	–	30
All	Trace element additives – other	–	10
All	Feed additives (binders and anti caking agents)	–	2
All	Premixes	–	15
Chromium			
Cattle, pigs, poultry, sheep	Total diet	–	20
Copper			
Cattle	Total diet	20 qw	20
Sheep	Total diet	20 qw	15
Pigs	Total diet	200 q, 220 w except breeders	100
Pigs for breeding	Total diet	50 qw	–
Chickens for meat	Total diet	200 q, 220 w	–
Chickens for breeding	Total diet	20 qw	–
Other animals	Total diet	20 qw	–
Cattle, goats, sheep	Stock licks	1400 qw	–
Fluoride			–
Dairy cattle, calves	Stock food	40 qw	40
Sheep, pigs (except breeding pigs)	Stock food	200 qw	200
Breeding pigs	Stock food	150 qw	150
Poultry	Stock food	350 qw	350
Cattle (except dairy), goats	Stock food	200 q	200
Dairy cattle	Lick or mineral supplement	400 q	400
Cattle (except dairy), goats, sheep	Lick or mineral supplement	2000 q	2000
All	Phosphate for inclusion in manufactured stock food	40[B] w	40
Lead			
Cattle, pigs, sheep	Total diet	0.2 nqw	5
All	Green fodder		30
All	Feed additives based on phosphates	1 w	15
All	Feed additives based on calcium carbonate	1 w	20
All	Feed additives based on zeolites of volcanic origin	1 w	60
All	Feed additives (binders and anti-caking agents except zeolites)	1 w	30
All	Premixes	1 w	200
All	Trace element additives based on zinc oxide	1 w	400
All	Trace element additives based on manganous oxide,	1 w	200
	iron carbonate or copper oxide	1 w	–
All	Trace element additives – other	1 w	100
Mercury			
Cattle, pigs, sheep	Total diet	0.02 nqw	0.01
All	Fish meal	0.4 nqw	–
Selenium			
Cattle, pigs, sheep	Total diet	–	0.3
Cattle, goats, sheep	Stock food other than licks or mineral supplements	0.1 q	–
Pigs, poultry	Stock food	0.3 q	–

Table 4. (*continued*)

Element, species	Feed	State maximum limits (mg/kg DM)	Recommended guidance maximum level (mg/kg DM)
Cattle, goats, sheep	Licks	1 q	–
Livestock (not camelids)	Manufactured stock food	1 w	–
Cattle, goats, sheep	Licks and premixes	5 w	–
Zinc			
Cattle, sheep	Total diet	–	50
Pigs	Total diet	–	250[C]

[A]Applies to Queensland (100 mg/kg of phosphorus in the stock food, Qld 1997) and Western Australia (the lower of 20 g/tonne phosphate or 100 g/tonne of phosphorus in phosphate for inclusion in manufactured stock food, WA 2006).
[B]Lower of 8 g/tonne of phosphate or 40 g/tonne of phosphorus in phosphate (WA 2006).
[C]Note young pigs can tolerate 3000 mg/kg (used post-weaning for prevention of diarrhoea).

interaction with other elements, age and species) (Suttle 2010). Concentrations are highest in kidney, liver and bone (NRC 2005).

Data on the lead content of several grains used as feed materials have been reported by the NRS (pers. comm). In most samples lead was not detected above the limit of reporting of 0.01 mg/kg for cereal grain, pulses and oilseeds. Little data are available publicly on the concentration of lead in premixes and mineral supplements sold in Australia. However, analyses of 100 premixes in the EU showed average lead concentrations of 19 mg/kg DM (EFSA 2004*b*). From a database of 198 samples of mineral supplements (EFSA 2004*b*), the average concentration was 3.4 mg/kg. Maximum values reported were in two samples of magnesium oxide with concentrations of 30 mg/kg.

Based on the Australian ML of 0.5 mg/kg WW for edible offal and a TF of 0.12 for cattle kidney (MacLachlan 2011), concentrations in the total diet should not exceed 0.5 ÷ 0.12 = 4.2 mg/kg DM. A level of 5 mg/kg DM should be adequate to ensure lead in kidney does not exceed 0.5 mg/kg WW. Codex has established a ML of 0.02 mg/kg for milk and using this value together with the TF reported by MacLachlan (2011) concentrations in the total diet of dairy cows should not exceed 0.02 ÷ 0.0024 = 8.3 mg/kg DM. As the level calculated for tissues (5 mg/kg DM) is lower than that calculated for milk (8 mg/kg DM), concentrations in the total diet should be as-low-as-reasonably-achievable and not greater than 5 mg/kg DM. Levels of lead in individual feed components should also be as-low-as-reasonably-achievable. Guidance levels for feed ingredients based on the results of the ESFA survey (EFSA 2004*b*) are summarised in Table 4.

Mercury

Mercury in the natural environment is found in both inorganic and organic forms (NRC 2005). Sources include some mineral deposits and from inclusion of fish meal in livestock diets (NRC 2005). The inorganic forms of mercury are less toxic than organic ones and among organic forms the most toxic is methyl mercury. Based on the FSANZ GEL of 0.01 mg/kg WW for edible offal of cattle, pigs and sheep and a TF of 4.6 for sheep kidney (MacLachlan 2011), concentrations in feed should not exceed 0.01 ÷ 4.6 = 0.002 mg/kg DM. If the calculation is repeated for cattle (GEL 0.01 mg/kg WW, TF kidney 0.89) the concentration in the total diet should not exceed 0.01 mg/kg DM. A level of 0.01 mg/kg DM should be adequate to ensure concentrations in kidney of livestock do not significantly exceed 0.01 mg/kg.

The proposed level of 0.01 mg/kg DM is lower than in the current state regulations listed in Table 4. Records of the original justification for regulated levels are not available for comparison. The level proposed here is estimated using the FSANZ GEL which in turn is based on the 90th percentile of mercury concentrations reported in surveys of Australian cattle, pig and sheep kidneys. As stated earlier, GELs are not regulatory standards but have been developed to assist food manufacturers in ensuring concentrations of contaminants are as-low-as-reasonably-achievable. Higher concentrations in feed may occur if fish meal is included in the ration though it is noted that in this case compliance with the FSANZ 90th percentile GEL might not be possible.

Selenium

Selenium exposure of livestock can be derived from consumption of selenium-accumulating plants (*Neptunia amplexicaulis* and *Morina reticulata*) growing on natural mineral outcrops in parts of Queensland (Tinggi 2003), and from its use as a supplement. The optimum nutritional requirements for selenium are uncertain. FSANZ has established a GEL for selenium concentrations in meat which can be used to derive a guidance level for feed. Based on a GEL of 0.2 mg/kg WW for meat of cattle, pigs and sheep and a TF of 0.72 for pig muscle (MacLachlan 2011), concentrations in the total diet should not exceed 0.2 ÷ 0.72 = 0.28 mg/kg DM. A level of 0.3 mg/kg DM should be adequate to ensure selenium does not exceed the GEL (90th percentile) value reported by FSANZ. Current state regulations are in Table 4. The level proposed for the total diet of 0.3 mg/kg DM agrees with the Queensland regulatory level of 0.3 mg/kg for pig and poultry stock food and is higher than the Queensland limit for cattle, sheep and goat stock food, but lower than the Western Australian level of 1 mg/kg for manufactured stock food.

Zinc

Sources of zinc include mineral deposits but are also widespread on farms, due to its use in galvanising metal used in structures

used in animal husbandry. It is an essential element added to diets of intensively raised livestock (Suttle 2010).

GEL values have been established by FSANZ for zinc in meat and offal and can be used to derive guidance levels for feeds. Based on a GEL (90th percentile) of 60 mg/kg WW for edible offal of cattle, pigs and sheep and TFs of 1.3 for cattle liver and 0.26 for pig liver (MacLachlan 2011), concentrations in feed should not exceed 60 ÷ 1.3 = 46 mg/kg DM for cattle and sheep and 60 ÷ 0.26 = 230 mg/kg DM for pigs. Levels of 50 mg/kg DM for cattle and sheep and 250 mg/kg DM for pigs should be adequate to ensure zinc does not exceed the GEL (90th percentile) proposed by FSANZ.

The guidance maximum levels do not take into account therapeutic use of zinc in the diet. Zinc is sometimes added to the total diet of young pigs post-weaning as a disease preventative at concentrations of up to 3000 mg/kg DM until a liveweight of 12 kg is reached (Jacela *et al*. 2010) and to cattle diets at up to 500 mg/kg DM for prevention of foot diseases and facial eczema (Dairy Australia 2011).

Both state and proposed guidance maximum levels for various elements are summarised in Table 4. The Supplementary Material (available on the Journal's website) contains a summary of the bioavailability of elements in different matrices relative to the forms used to estimate the transfer from feed to tissues, milk and eggs.

Phytotoxins

Phytotoxins are defined here as toxic substances naturally produced by plants hence they are regarded as 'natural toxins'. Most have evolved with the plants as defences against insects and other predators, but a small proportion are toxic to livestock (Barry and Blaney 1987), and also present some risk of producing residues in livestock products. Feed ingredients from grain and pulse crops can contain their own respective natural toxins, and can also contain others derived from weeds growing within the crop or at field margins at the time of harvest. Examples of undesirable substances that may be produced as natural components of a crop include gossypol production by cotton (*Gossypium* spp.) and tetrahydrocannabinol (THC) production by hemp (*Cannabis sativa*). Fodder crops such as rye grass (*Lolium* spp.) can also contain simple toxins, like nitrate and oxalate, which can poison livestock but are not persistently residual in livestock products. Advice is freely available to producers to manage the risk of nitrate and oxalate poisoning in grazing livestock, but risks remain for purchased hays and preserved fodder.

Gossypol

Gossypol in cottonseed meal or oil is transferred to edible tissues including muscle and offal of ruminants and poultry, as well as into eggs and milk (EFSA 2008). Available information on transfer from feed suggests the maximum levels for feed that apply in the EU are adequately protective of livestock and also limit gossypol concentrations in food (EC 2003; EFSA 2008).

Nitrates

Under normal conditions the nitrate ingested by ruminants is converted to ammonia and then bacterial protein in the rumen (EFSA 2009). Nitrites are formed as an intermediate in the conversion of nitrate to ammonia. The conversion of nitrates to nitrite occurs at a faster rate than nitrite is converted to ammonia. Consequently, when higher than normal amounts of nitrate are consumed, an accumulation of nitrite may occur in the rumen. Nitrite then will be absorbed into the bloodstream and will convert hemoglobin to methemoglobin, which is unable to transport oxygen, leading to nitrate poisoning (Hill and Blaney 1980; Bruning-Fann and Kaneene 1993). High concentrations of nitrates result in depressed feed intake. Toxic levels of nitrogen in the form of nitrate are above 5000 mg nitrogen/kg DM in the diet (EC 1978), although this varies up or down with the condition of the livestock. A guidance level of 2000 mg nitrogen/kg DM (0.2%) is proposed, but expressed as the potassium nitrate equivalent of 10 000 mg/kg (1%).

Oxalate

Exposure of livestock to oxalates present in plant material may lead to the precipitation of insoluble calcium oxalate in the kidneys leading to acute renal failure, or in the longer term to calcium deficiency (Blaney *et al*. 1981, 1982; Rahman *et al*. 2012). Poisoning in sheep and cattle has been reported when pasture contains 7–8% soluble oxalate on a DM basis (Seawright *et al*. 1970; James *et al*. 1971; James and Butcher 1972) while the presence of 3% oxalates in the diet of lambs significantly depressed voluntary feed intake (Burritt and Provenza 2000). McKenzie *et al*. (1988) reported that levels of 2% or more soluble oxalate (sodium and potassium oxalates, expressed as potassium oxalate equivalent) can lead to acute toxicosis in sheep. A guidance level of 10 000 mg soluble oxalates (expressed as potassium oxalate)/kg DM (1%) should provide a sufficient margin of safety for livestock exposed to feed containing oxalates.

Tetrahydrocannabinol

THC is the main psycho-active compound found in the leaves and flowering heads of *C. sativa* and is a fat-soluble compound. If hemp products are fed to livestock, THC may transfer to milk and fat. In Queensland, the *Stock Regulation 1988* (Qld 1988) imposes restrictions on feeding hemp (*C. sativa*) to stock. Under the *Stock Regulation 1988* (Qld 1988) only low THC containing cannabis products (processed cannabis, oil extracted from processed cannabis and meal ground from processed cannabis) are permitted to be fed to stock. In New South Wales the *Stock Foods Regulation 2010* prohibits the inclusion of stock feed of all cannabis plant parts other than seeds which have had the outer bracts removed.

EFSA have recently reviewed available information on the use of industrial (low THC) hemp as a livestock feed and the transfer of THC from hemp to food of animal origin (EFSA 2011). The EFSA Panel on Additives and Products or Substances used in Animal Feed recommended feeds derived from whole hemp plants should be either restricted or prohibited due to the potential for transfer of unacceptable levels of THC and related compounds into food of animal origin. Consistent with the Queensland Stock Regulation, they noted that hemp seed contains lower levels of THC and related compounds compared with whole plants and that hemp seed-derived feed ingredients should not contain more than

10 mg THC/kg DM when included at a maximum of 20% of the total diet.

Other plant toxins

A large number of potentially toxic plants may contaminate pasture and feed (Cheeke 1998). In Australia, Paterson's curse (*Echium plantagineum*), perennial ryegrass (*Lolium perenne*), *Pimelea* spp. and St John's wort (*Hypericum perforatum*) are a few out of many that are commonly recognised as poisonous plants weeds of importance affecting grazing livestock. An extensive investigation during 2002–08 assessed the risks to human health and trade from the transfer of natural toxin residues from rangeland plants and seeds to foods of animal origin (Blaney *et al*. 2008*a*). A comprehensive review of all of the 92 different toxins and toxin groups known to affect animals in Australia and overseas, identified only a very few that were considered as having potential to produce concentrations in foods of animal origin of possible human health significance. These were: pyrrolizidine alkaloids [rattlepods (*Crotalaria* spp.), heliotropes (*Heliotropium* spp.), fireweeds (*Senecio* spp.), Paterson's curse, and others]; indospicine [Birdsville indigo, creeping indigo, etc (*Indigofera* spp.)]; and ptaquiloside [bracken fern (*Pteridium* spp.), mulga fern (*Cheilanthes* spp.)]. Further detailed investigation concluded that the risk to health of persons consuming meat from livestock exposed to pyrrolizidine-containing plants in northern Australia was negligible (Fletcher *et al*. 2011*a*). The risks from indospicine in meat and ptaquilosides in milk are not considered high, but are still under investigation (Fletcher *et al*. 2011*b*). However, most exposure to the source plants is from grazing situations rather than from contaminated feedstuffs. Consequently, with the current state of knowledge, guidance levels for these natural toxins in feeds are not considered necessary but will be re-assessed as further data become available.

Weed seeds

Weed seeds can have two potential impacts as contaminants of feed grains. First, toxic components within the seeds, or mechanical irritation caused by seeds such as burrs, can affect livestock health and productivity. Second, the use of weed-contaminated grains in livestock feeds can spread the weed and exacerbate the weed problem. When assessing the consequences of weed seed contamination from a livestock perspective, the biggest consideration is any natural toxin that the weed seed contains. If a weed seed does not contain a toxin, its presence in a grain sample may be of limited concern except for the effect it may have on the ability to define the supply of amino acids and energy that could be expected from the grain sample in question.

A review of Australian weeds (R. A. McKenzie, unpubl. data) identified the following plants as responsible for adversely affecting livestock production when their seeds contaminate feed: Mexican poppy (*Argemone mexicana* and *A. ochroleuca*), jute (*Corchorus olitorius*), rattlepods *Crotalaria* spp., thornapples (*Datura* spp.), Paterson's curse (*E. plantagineum*), common heliotrope (*Heliotropium europaeum*) and blue heliotrope (*H. amplexicauli*), bellvine (*Ipomoea plebia*), darnel (*Lolium temulentum*), annual yellow sweet clover (*Melilotus indicus*), climbing buckwheat (*Polygonum convolvulus*), castor oil plant (*Ricinus communis*), sesbania pea (*Sesbania cannibina*), caltrop (*Tribulus terrestris*), common vetch (*Vicia sativa*) and burrs (*Xanthium* spp.). The toxins contained in the seeds include pyrrolizidine alkaloids (rattlepods, Paterson's curse, heliotropes), ricin (castor oil plant), dihydrosanguanarine and dihydrochelerythrine (Mexican poppy) and scopolamine (thornapples). On the basis of plant prevalence, consumption by livestock and persistence of levels in tissues, none of these toxins was considered to present a significant risk of meat contamination except for pyrrolizidine alkaloids and after further investigation that risk is now considered low (Blaney *et al*. 2008*a*).

Grain Trade Australia (GTA 2011) nominates weed and foreign seed levels in its purchasing standards with tolerances listed for contamination for a total of 192 weed seeds. Some weed seeds prohibited by state laws against inclusion in stock feeds such as castor oil plant and rattlepods, are listed by GTA as having 'nil' acceptance levels. Others listed above, that are known to be potentially toxic to livestock, are restricted to levels ranging from 2 to 400 seeds/half litre (0.002–0.2%), according to relative toxicity of the different seeds. Although exact tolerances by different livestock are not known, the restrictions appear to offer a generous safety margin below known toxic levels (Spencer 2010). Sufficient information is not available to estimate guidance levels for most weed seeds. Seed contamination of grain to protect livestock health is best handled through existing GTA receival standards (GTA 2011).

Proposed guidance maximum levels for phytotoxins are summarised in Table 5.

Mycotoxins

Mycotoxins are 'natural toxins' produced by fungi, which have evolved as defences against predators and competitors, and which are toxic to animals (Blaney 1996). Mycotoxin contamination of forages, cereals and pulse crops frequently occurs in the field following infection of plants with particular pathogenic fungi or with symbiotic endophytes (D'Mello 2004). Production of mycotoxins by fungi can also occur during processing and storage of harvested feed materials when environmental conditions, particularly moisture and ambient temperature are appropriate for development of spoilage fungi (D'Mello 2004). A wide range of mycotoxins affect livestock health in Australia, as in other countries (Bryden 2012*a*). Most are metabolised fairly quickly after ingestion, and residues do not appear to accumulate or persist in animal products for long periods (Bryden 2009; MacLachlan 2011). Sufficient concerns have been raised regarding aflatoxins in milk and offal, and ochratoxin A in meat and meat products (particularly pork and veal), to warrant regulation in various countries. It should be noted that human exposure to these mycotoxins is predominantly from direct ingestion of contaminated food crops, with exposure from consumption of livestock products relatively minor (Miller 2008; FSANZ 2011).

The 20th Australian Total Diet Survey (FSANZ 2003) investigated the dietary exposure of the Australian population to aflatoxins and ochratoxin A. Aflatoxins (B1, B2, G1 and G2) and ochratoxin A were not found in any food tested, namely: breads, biscuits, rice, oats, processed wheat bran, breakfast

Table 5. State and recommended guidance maximum levels for phytotoxins in feed

Phytotoxin	Feed	Guidance maximum level (mg/kg DM)
Gossypol	Total diet adult cattle, goats and sheep	500
	Total diet calves and poultry other than laying hens	100
	Total diet for pigs other than piglets	60
	Total diet for piglets and laying hens	20
	Cotton seed	5000
	Cotton seed cakes and meal	1200
	other feed materials	20
Nitrates (as potassium nitrate)	Total diet cattle, goats, pigs and sheep	10 000
Oxalates (as potassium oxalate)	Total diet cattle, goats, pigs and sheep	10 000
Tetrahydrocannabinol	Hemp seed-derived feed ingredients	10 (and max. 20% of the diet)

cereals (including infant cereal), instant coffee, peanut butter, almonds and milk chocolate. More recently the 23rd Total Diet Survey (FSANZ 2011) did not detect aflatoxins (B1, B2, G1, G2 and M1), deoxynivalenol (DON), fumonisins (B1 and B2), ochratoxin A, patulin or zearalenone (ZEA) in any food analysed. Mycotoxins other than these have not been considered to be public health concerns at the concentrations so far reported in Australian food crops.

Table 6 lists MLs for food of animal origin for various mycotoxins and includes levels for major markets for Australian livestock commodities.

It is evident that few MLs have been established for mycotoxins in foods of animal origin (Table 6). In many cases the concerns over the presence of mycotoxins in feed relate to their effects on production rather than the safety of animal-derived foods (Bryden 2012*b*). The mycotoxins relevant to livestock production are aflatoxins, ochratoxin A, fumonisins, ZEA, trichothecenes and ergot alkaloids (Bryden 2012*b*), while aflatoxins, ochratoxin A and ZEA are also of concern for food of animal origin (see Table 7). In considering guidance levels for mycotoxins, their transfer to animal commodities as well as the effect of feed concentrations on production are considered. The guidance levels proposed here are generally below the lowest adverse effect levels reported in the literature for the various livestock species (Eriksen and Pettersson 2004; Pettersson 2004). It is noted that a few additional mycotoxins relevant to feed have either been detected in Australia, or are likely to occur here based on fungal prevalence, including moniliformin, cyclopiazonic acid, sterigmatocystin, citrinin, alternariols, tenuazonic acid, patulin, etc. Based on current knowledge, these are not considered to present significant risks to either livestock health or to contamination of animal products (B. J. Blaney, unpubl. data), nor are guidance levels considered to be warranted.

Table 6. Maximum limits and action levels (μg/kg wet weight) applicable to mycotoxins in meat, meat products and milk in various countries and regions

Mycotoxin	Meat and meat products	Milk	Country/region	Reference
Aflatoxin M1	–	0.5	Brazil	Brazil (2011)
Aflatoxin M1	–	0.05	Chile	Chile (2011)
Aflatoxin M1	–	0.5	China	China (2011)
Aflatoxin M1	–	0.5	Codex	Codex (2010)
Aflatoxin M1	–	0.05	EU	EC (2006*b*)
Aflatoxin M1	–	0.5	Korea	Korea (2011)
Aflatoxin M1	–	0.5	Russia	Russia (2010)
Aflatoxin M1	–	0.5	Taiwan	Taiwan (2009*d*)
Aflatoxin M1	–	0.5	USA	USFDA (2011)
Aflatoxin B1 + B2 + G1 + G2	30[A]	–	Brazil	Brazil (2011)
Aflatoxin B1 + B2 + G1 + G2	5[A]	–	Chile	Chile (2011)
Aflatoxin B1	20[A]	–	USA	USFDA (2011)
Aflatoxin B1 + B2 + G1 + G2 + M1 + M2 + aflatoxicol	15[A]	–	Hong Kong	Hong Kong (2011)
Aflatoxin B1	ND[AB]	–	Japan	Japan (2011*b*)
Ochratoxin A	25 (10) pork[C]	–	Denmark	FAO (2004)
Zearalenone	200[A]	–	Chile	Chile (2011)

[A]Maximum levels are for all food and would include meat and meat products.
[B]ND = no detection permitted, limit of detection for the reference method is 10 μg/kg wet weight.
[C]Kidney levels: 25 μg/kg condemn pig carcass; 10 μg/kg condemn pig kidney.

Table 7. Recommended guidance maximum levels for mycotoxins in feed
n = New South Wales; q = Queensland; w = Western Australia

Mycotoxin	Feed	State maximum limit (mg/kg DM)	Recommended guidance maximum level (mg/kg DM)
Aflatoxins (B1 + B2 + G1 + G2)	Ducks	0.001[A] nqw	0.002
	Layer chickens	0.02[A] nqw	0.03
	Poultry (other than ducks, layer chickens)	0.01[A] nqw	0.02
	Weaner pigs	0.01[A] nqw	0.02
	Grower pigs, finisher pigs	0.05[A] nqw	0.08
	Beef cattle, sheep	0.05[A] nqw	0.08
	Dairy cows, dairy sheep, dairy goats	0.02[A] nqw	0.03
	Feed ingredients		–
	Cotton seed, peanut meal, peanut screenings	0.2[A] nw	–
	Meal of canola, coconut, linseed, lupin, pea, safflower, soybean and sunflower	0.1[A] nw	–
	Grain, crushed	0.01[A] nw	–
Ochratoxin A	Calves, kids, lambs, weaner pigs	–	0.05
	Pigs (other than weaner pigs)	–	0.1
	Poultry	–	0.1
	Cattle, sheep, goats	–	0.2
Fumonisins (B1 + B2 + B3)	Weaner pigs	–	5
	Grower pigs, finisher pigs	–	10
	Calves, kids, lambs	–	10
	Layer and breeder poultry	–	15
	Dairy and breeding cattle, sheep, goats	–	15
	Meat chickens, turkeys, ducks	–	30
	Cattle, sheep, goats	–	30
Zearalenone	Breeding sows, weaner pigs	–	0.05
	Dairy and breeding cattle, sheep, goats	–	0.2
	Calves, kids, lambs	–	0.5
	Grower pigs, finisher pigs	–	2
	Non-breeding cattle, sheep, goats	–	2
	Poultry	–	2
Deoxynivalenol + nivalenol (deoxynivalenol + nivalenol + their acetyl derivatives)	Pigs	–	0.5
	Calves, kids, lambs	–	1
	Cattle, sheep, goats, other than young animals	–	2
	Poultry	–	2
Rye ergots (rye ergot[B] or total rye ergot alkaloids[C] in the total diet)	Cattle, sheep, goats	200 (0.02%) as ergot qw	100 (0.01%) as ergot or 0.2 mg alkaloids/kg DM
	Weaner pigs, breeder pigs	200 (0.02%) as ergot qw	200 (0.02%) as ergot or 0.4 mg alkaloids/kg DM
	Grower pigs, finisher pigs	200 (0.02%) as ergot qw	1000 (0.1%) as ergot or 2 mg alkaloids/kg DM
	Poultry	200 (0.02%) as ergot qw	2000 (0.2%) as ergot or 4 mg alkaloids/kg DM
Ergot other than sorghum ergot	All species	200 (0.02%) as ergot qw	–
Sorghum ergot (sorghum ergot[D] or total sorghum ergot alkaloids[E] in the total diet)	Dairy and breeding cattle, goats, sheep	–	300 (0.03%) as ergot or 0.1 mg alkaloids/kg DM
	Non-breeding cattle, sheep, goats	–	1000 (0.1%) as ergot or 0.3 mg alkaloids/kg DM
	Weaner pigs, breeder pigs	–	1000 (0.1%) as ergot or 0.3 mg alkaloids/kg DM
	Grower pigs, finisher pigs	–	10 000 (1%) as ergot or 3 mg alkaloids/kg DM
	Poultry	–	20 000 (2%) as ergot or 6 mg alkaloids/kg DM
	All species	3000 (0.3%) as ergot qw	–

[A]Aflatoxin B1 only. [B]The sclerotia of *Claviceps purpurea*. [C]The total of ergotamine, ergocryptine, ergosine, ergocornine, ergocristine and their respective C8 epimers, ergotaminine, ergocryptinine, ergosinine, ergocorninine and ergocristinine; or if only ergotamine is measured, 3 × ergotamine. [D]Sorghum ergot, *Claviceps africana*, is defined as the total weight of all ergot bodies, including mature sclerotia, sphacelia with glumes attached, and sphacelia overgrown with sporodochia of the ergot saprophyte *Cerebella* spp. [E]The total of dihydroergosine, dihydroelymoclavine and festuclavine or if only dihydroergosine is measured, 1.2 × dihydroergosine.

Strategies to mitigate the effect of mycotoxin contamination of feed and the effects on livestock production exist (Jard *et al.* 2011). Feed can be subject to decontamination treatments or the mycotoxin detoxified. The most common approaches reported are the inclusion of sorbent materials in the feed to reduce the amount of toxin absorbed from the gastrointestinal tract and the use of de-toxifying enzymes or microorganisms (Jard *et al.* 2011). The effectiveness of treatments in practical situations has not always been adequately demonstrated (Kolossova *et al.* 2009). The guidance levels proposed here (Table 7) assume mitigating treatments have not been employed before use of contaminated feed.

Aflatoxins

Aflatoxins are a group of chemically similar compounds produced mainly by *Aspergillus flavus* and *A. parasiticus*. The principal aflatoxins are B1, B2, G1 and G2. All four aflatoxins can contaminate livestock feed though B1 is the most toxic. Feeds most susceptible to aflatoxin are summer cereals (especially maize), cottonseed, peanuts and copra meal.

Aflatoxins are best known in Australia as a problem in rain-fed (non-irrigated) peanuts grown in parts of the Burnett region in south-east Queensland (Graham 1982; Blaney 1985; Rachaputi *et al.* 2002), but are also a problem in maize for similar reasons. *A. flavus* is able to grow in peanuts and maize of lower moisture content and at higher temperatures than many other fungi found on field crops. In healthy peanuts and maize, plant defences prevent growth of *Aspergillus* spp., but when low available moisture and high temperatures affect kernel development, plant defences are lowered and these fungi can invade. Maize is the only grain crop where aflatoxins are known to cause serious pre-harvest contamination. Significant concentrations of aflatoxins have not been detected in wheat and barley (or grain sorghum) without a history of storage problems. Cottonseed, other oilseeds and nuts like pistachios appear to have minor contamination problems in Australia compared with elsewhere. Low concentrations might occasionally be detected in mouldy hay and straw, but generally *A. flavus* and *A. parasiticus* are not competitive with other fungi on these high-cellulose materials.

There are reports of livestock being affected by aflatoxins in Australia including chickens (Gardiner and Oldroyd 1965), turkeys (Hart 1965), ducks (Bryden *et al.* 1980), pigs (Ketterer *et al.* 1982) and cattle (McKenzie *et al.* 1981). The sources of aflatoxin in these cases included peanut meals and by-products, mouldy bread and other bakery waste, and grain sorghum and maize that were stored with high moisture contents.

Although Queensland (Qld 1997) has regulated aflatoxins in feed for many years to protect animal health, the need for this is under review, particularly as the peanut and maize industries (those with most aflatoxin problems), have a long history of responsible mycotoxin management in their stock feeds and by-products. Nevertheless, there remains strong justification for regulating aflatoxin concentrations in feed to minimise resultant milk contamination. Aflatoxin B1 is converted to aflatoxin M1 in the mammalian liver and ~1–6% of aflatoxin B1 ingested by lactating animals is transmitted into milk as aflatoxin M1. The USA has a limit of 0.5 µg/L (USFDA 2011) for aflatoxin M1 in milk while the EU has a limit of 0.05 µg/L (EC 2006*b*). The EU limit of 0.05 µg/L is not consistent with widely applied feed limits, including its own limit of 0.02 mg/kg (EFSA 2004*c*). For example, based on an ML of 0.05 µg/L for milk and a TF of 0.015 for cattle milk (MacLachlan 2011), levels of aflatoxin B1 in the total diet of dairy cattle should not exceed 0.00 005 ÷ 0.015 = 0.0033 mg/kg DM. If bulking and blending of milk from individual cows occurs before use a greater level in feed could be accommodated. Some countries have established MLs for food in general and based on an ML of 5 µg/kg WW for food (includes meat and meat products) in Chile together with a TF of 0.0025 for cattle liver (MacLachlan 2011), levels of aflatoxin B1 in the total diet of beef cattle should not exceed 0.005 ÷ 0.0025 = 2 mg/kg DM. However, this would be a toxic concentration for beef cattle, which can tolerate only 0.1–0.3 mg/kg DM over a short term. We therefore recommend a guidance limit of 0.08 mg/kg DM, which, using the TF of 0.0025 for alfatoxin B1 and assuming the TF also applies to the sum of aflatoxins B1 + B2 + G1 + G2, could produce a maximum aflatoxin concentration of 0.2 µg/kg in liver.

Aflatoxins are carcinogens and residues in food should be as-low-as-reasonably-achievable (IARC 1993). There is no evidence that the current Australian maximum levels in state legislation for feed are inappropriate. Concentrations of aflatoxin in feed should not be higher than permitted by existing standards. To enable comparison with those standards specified in terms of the sum of aflatoxins B1 + B2 + G1 + G2 rather than aflatoxin B1, existing standards in the Queensland regulations (Qld 1997) have been converted into the most likely equivalent concentrations of total aflatoxins B1 + B2 + G1 + G2 in feeds, total aflatoxins = 1.6 × aflatoxin B1 (Weidenbörner 2001). Although chickens, adult ruminants and finisher pigs may tolerate much higher concentrations (0.1–0.3 mg/kg DM) for short periods, it is considered that the Australian grain and feed industries can meet the lower levels with good agricultural practice. The maize industry has set its own limit of 0.08 mg/kg DM for Feed No. 2 grade. Guidance maximum levels for total aflatoxins B1 + B2 + G1 + G2 in the total diet are listed in Table 7.

Ochratoxin A

Ochratoxin A is produced by *Aspergillus ochraceus*, *A. carbonarius*, *A. niger* and *Penicillium verrucosum* (Pitt *et al.* 2000). Ochratoxin contamination has been identified in grapes and grape products in Australia, mainly due to *A. carbonarius* (Hocking *et al.* 2003), and is known as a contaminant of coffee beans and figs elsewhere. Growth of *P. verrucosum* in storage has caused serious ochratoxin contamination of barley and other grains in Canada and Europe, but it is generally not found in NRS surveys of Australian small grains (NRS, unpubl. data) nor during screening of several hundred ‘fair average quality’ sorghum samples from Queensland (B. J. Blaney, unpubl. data). Ochratoxin A has been detected on rare occasions and in very low concentrations (0.001–0.004 mg/kg) in maize at harvest in Australia however, most surveys of Australian maize have not detected any ochratoxin A (Blaney *et al.* 1984*b*, 1986; Bricknell *et al.* 2008; R. Maryam and B. J. Blaney, unpubl. data). There have been no proven cases of ochratoxin poisoning of cattle, goats, sheep, pigs or poultry in Australia.

In ruminants, ochratoxin A is degraded by rumen protozoa and bacteria to the less toxic ochratoxin-α with negligible transfer of ochratoxin A to tissues and milk (Müller *et al*. 1998). Ochratoxin A is widely distributed in monogastric species such as pigs and chickens with highest concentrations observed in kidney and liver. Denmark has implemented a management system for pigs where kidneys with concentrations above 0.01 mg/kg WW are discarded. The TF estimated by MacLachlan (2011) was 0.11 for kidney and ochratoxin A in the total diet for pigs should not exceed 0.01 mg/kg ÷ 0.11 = 0.1 mg/kg DM to meet the management level implemented in Denmark.

Currently, there are no regulations for ochratoxin A in Australia and all indications are that it does not present a significant risk to human health or livestock in Australia. The very low contamination in Australian feed does not justify any specific restriction on feed nor regulatory levels for food of animal origin to protect human health. Guidance for animal feed could be justified in light of the need to address concentrations that might occur in imported feed, feed ingredients and feed additives. Additionally, the increasing scrutiny of feed materials using increasingly sensitive assay methods is likely to lead to more detection, which will raise problems in interpreting risk to livestock unless levels are available to interpret their significance.

Pigs have been found to be the food-producing animals most sensitive to ochratoxin A. Microscopic lesions were detected in the kidney of female pigs fed 1 mg/kg feed for 2 years (Kuiper-Goodman and Grant 2007) and reduced growth rates reported for pigs fed 0.2–2 mg/kg feed (Madsen *et al*. 1982*a*, 1982*b*; Tapia and Seawright 1984). Ruminants are less sensitive due to conversion of ochratoxin A to ochratoxin-α in the rumen and levels in the total diet of 0.2 mg/kg DM for ruminants and 0.1 mg/kg DM for adult pigs should be adequately protective. A concentration of 1 mg/kg has been shown to produce minor kidney damage in long-term feeding studies with young pigs, so 0.05 mg/kg DM in the total diet would appear to offer a generous safety margin when applied to young pigs and pre-ruminant animals (calves, kids and lambs). Australian broiler chickens (3 weeks of age) fed 1 mg/kg for 5 weeks were not adversely affected (Reichmann *et al*. 1982). Other studies have reported reduced growth and also reduced egg production in chickens exposed to 0.5–1 mg/kg feed (Huff *et al*. 1975; Krogh *et al*. 1976; Prior and Sisodia 1978; Page *et al*. 1980). A guidance level of 0.1 mg/kg DM is suggested for poultry. Guidance maximum levels for ochratoxin A in the total diet are proposed and listed in Table 7. The suggested levels have substantial safety margins in relation to known no observable effect levels for livestock reviewed by EFSA (2004*d*), but available survey data suggest that meeting these levels should have minimal impact on Australian feed producers and are consistent with the as-low-as-reasonably-achievable principle.

Fumonisins

Fumonisins are a group of chemically related polar compounds based on a hydroxylated hydrocarbon chain with methyl and amino (or acetyl) substituents (CAST 2003). The most common and toxic is fumonisin B1, with B2 and B3 usually accompanying B1 but in much lower concentrations (Marasas 1996). Fumonisins appear predominantly to occur in maize, being produced by several *Fusarium* spp. that are associated with ear rot and stalk rot in maize worldwide with some reports also in sorghum (Munkvold and Desjardins 1997; Leslie and Marasas 2001). The most common species is *Fusarium verticillioides* (previously called *F. moniliforme*, Seifert *et al*. 2003), which appears to be the main source of fumonisins (Munkvold and Desjardins 1997). Fumonisins are present in maize in all Australian growing regions and concentrate in lightweight grain screenings (Bricknell *et al*. 2008).

In Queensland, the significance of kernel-rot varies between seasons. For example, it was severe on the southern Downs in 1985–86 (Williams *et al*. 1992) and some concentrated samples of damaged kernels obtained during 1985–86 were later found to contain up to 40 mg fumonisins/kg (B. J. Blaney, unpubl. data). In 2003, maize grown in the Murrumbidgee Irrigation Area of southern New South Wales had an increase in kernel-rot, and several samples contained 5–50 mg fumonisins/kg (Blaney *et al*. 2008*b*). However, concentrations of fumonisins in maize from surveys conducted of Australian maize-growing regions in 2004–06 were >0.1 mg/kg in 66% of samples with over 85% of all samples complying with the GTA standard for milling grade maize (2 mg/kg) (Bricknell *et al*. 2008). Only 2 of 567 samples exceeded the GTA grade 2 feed standard (40 mg/kg).

Fumonisins are not currently regulated by governments in Australia. The GTA trading standards set by the maize industry for the sum of fumonisins B1 + B2 + B3 are 10 mg/kg for GTA grade 1 feed and 40 mg/kg for GTA grade 2 feed and are consistent with overseas proposals (USFDA 2001*a*, 2001*b*). The risks for livestock appear low given that maize is rarely the main grain component of mixed diets in Australia.

Studies on the carry-over of fumonisins from feed into animal products all indicated that low concentrations of fumonisin B1 can be detected in various tissues, but the low rate of transfer suggests animal products do not contribute substantially to human exposure (EFSA 2005*a*; MacLachlan 2011).

In animals, fumonisins (particularly B1) are known to cause a wide range of illnesses such as leucoencephalomalacia in horses (Shanks *et al*. 1995) and pulmonary oedema in pigs. The experimental oral dose leading to porcine pulmonary oedema after less than 5 days exposure is 20 mg/kg bodyweight per day (Gumprecht *et al*. 2001), while a dose of 0.4 mg/kg bodyweight per day was sufficient to cause mild cases in piglets when fed to pigs for a period of 4 weeks (Zomborszky *et al*. 2000).

The guidance levels listed in Table 7 are consistent with the United States Food and Drug Administration (USFDA 2001*b*) guidelines and also in harmony with the maize industry standards for feed grade maize once maize is formulated into complete diets (GTA 2011).

Zearalenone

ZEA is a substituted resorcyclic acid lactone. It is a non-steroidal estrogenic mycotoxin that has been implicated in some forms of infertility in pigs, cattle, sheep and possibly other animals. It has not been proven to affect human health (EFSA 2004*e*).

In maize, wheat, barley and triticale, ZEA is primarily produced by *F. graminearum*, a fungus responsible for causing ear and stalk rots of maize and head scab (head blight) of small grains (Burgess *et al*. 1981). Other species known to

produce ZEA in grain include *F. culmorum*, *F. equiseti* and *F. crookwellense*, while *F. pseudograminearum* is a major source in wheat and barley crowns and stalks.

In maize, significant concentrations of ZEA can be most frequently detected on the tablelands of Far North Queensland where the persistently wet climate favours ear rot by *F. graminearum* and concentrations of ~1 mg/kg can be quite common (Blaney *et al*. 1984*b*, 1986). The most contaminated kernels can be distinguished by a dark purplish colouration. Samples with 2% of purple kernels contained ~1 mg ZEA/kg. Continued breeding of maize hybrids for resistance to ear rot appears to have decreased ZEA concentrations over the last 20 years. Another area where *F. graminearum* is relatively common in maize is in the wet coastal districts of south-east Queensland and northern New South Wales. ZEA contamination in these zones is related to the presence of inoculum but incidence is determined by timing of rainfall in relation to silking and the relative resistance of the maize hybrids planted. In wheat, barley and triticale, head blight caused by *F. graminearum* and *F. culmorum* occurs at quite low prevalence, even in wetter regions of Australia where maize (and sorghum) is also grown. A survey of all wheat grown in south-east Queensland in 1983–85 by Blaney *et al*. (1987) reported a maximum level of 0.04 mg/kg in bulk wheat with a maximum of 0.43 mg/kg in an individual wheat delivery to a storage facility.

Factors favouring *F. graminearum* infection are the key to the difference in prevalence of ZEA in maize compared with the small grains. *F. graminearum* causes head blight of wheat, and rotating wheat and maize is a common cause of increased infection in both crops if climatic factors suit (Blaney *et al*. 1987; Southwell *et al*. 2003). Head blight is occasionally detected in other areas of Australia when there are unusually wet springs. Because of climate and limited production of maize in wheat-growing regions, it can be concluded that the risk of ZEA contamination of wheat grain is low in Australia. In contrast, the risk of ZEA contamination of hay prepared from wheat and barley stalks is high. *F. pseudograminearum* is the cause of crown rot of wheat and barley in Australia, one of the most serious diseases of wheat in the northern wheat-growing regions. This fungus produces very high ZEA concentrations (20–40 mg/kg) in the crowns and stalks of infected plants (Blaney *et al*. 1987). ZEA is also a common contaminant of weather-damaged sorghum grain in Queensland, though usually at low concentrations. Storage of weather-damaged sorghum in moist, cool conditions can allow high concentrations to develop. There is a growing body of evidence that Fusarium species capable of ZEA production are also widespread in Australian pastures (Reed and Moore 2009).

There is only limited transmission of ZEA and its oestrogenic metabolites into tissues, milk and eggs (MacLachlan 2011). EFSA (2004*e*) concluded that due to rapid biotransformation and excretion of ZEA in animals, that secondary human exposure from residues in milk, meat and eggs was expected to be low and contribute only marginally to the daily intake. Chile has established an ML for food, and based on the ML of 200 µg/kg for food in Chile and a TF of 0.021 for muscle of pigs (MacLachlan 2011), levels in the total diet should not exceed $0.2 \div 0.021 = 9.5$ mg/kg DM. Animal production considerations require lower levels in feed.

A concentration of 8 mg/kg ZEA was detected in sorghum grain used as feed and associated with oestrogenic effects in pigs on the Atherton Tableland (Blaney *et al*. 1984*a*). Young female pigs appear to be the most susceptible class of animal to ZEA, where oestrogenic effects can be produced at concentrations of 0.2 mg/kg in the total diet although these effects are more consistent at 1 mg/kg (Williams *et al*. 1988; Williams and Blaney 1994). In some field cases, effects are associated with even lower concentrations (0.1 mg/kg) and it appears that once young pigs have ingested sufficient ZEA to induce pseudo-oestrogenism that lower concentrations can maintain that condition (Biehl *et al*. 1993). Guidance levels of 0.05 mg/kg DM are proposed for young pigs and also breeding sows.

Ewes are also sensitive to ZEA (Smith and Morris 2006). Studies by Smith *et al*. (1990) showed that doses of ZEA equivalent to dietary concentrations of ~1 mg/kg administered from Day 7 of oestrus until mating reduced ovulation rate, increased duration of oestrus and increased uterine weight. Higher concentrations reduced incidence of ovulation and reduced fertilisation. ZEA given several days after mating did not produce adverse effects (Smith *et al*. 1990).

New Zealand pasture samples associated with reduced ewe fertility have been recorded as containing 0.2–2.6 mg/kg DM (di Menna *et al*. 1985). The susceptibility of young heifer cattle has not been adequately explored but a similar tolerance as ewes might be expected. In New Zealand, reproductive problems in dairy cattle have been associated with dietary concentrations of ~0.4 mg/kg (Towers *et al*. 1995). Guidance levels of 0.2 mg/kg DM are proposed for dairy and breeding ruminants and 0.5 mg/kg DM for young ruminants. Non-breeding cattle, goats, sheep and pigs can tolerate much higher concentrations and a guidance level of 2 mg/kg DM is proposed for these animals.

Poultry are quite resistant to ZEA and concentrations ranging from 10 to 800 mg/kg have been required to produce significant effects (Chi *et al*. 1980*a*, 1980*b*; Allen *et al*. 1981*a*, 1981*b*; Olsen *et al*. 1986; Maryamma *et al*. 1992). A guidance level of 2 mg/kg DM would offer a considerable safety margin.

Fortunately the low prevalence of ZEA contamination in Australian feeds, with the notable exception of wheaten/barley hays and possibly pasture hays, allows a safety margin to be used in proposing guidance levels. The guidance levels listed in Table 7 allow for individual feed ingredients such as maize to contain 0.1–2 mg/kg DM and on current information >99.9% of Australian maize would meet this standard.

Trichothecenes

DON and nivalenol (NIV) are trichothecenes, a large group of sesquiterpenes that are broadly divided into Type A (T-2 toxin; HT-2 toxin, diacetoxyscipenol, etc), Type B (DON, NIV) and macrocyclic trichothecenes (verrucarins, roridins). Acute exposure to trichothecenes induces anorexia at low doses and emetic effects at higher doses as well as causing problems with cell replication, irritation of the gastrointestinal tract and effects on the immune system. Type A trichothecenes including T-2 toxin produced by *F. sporotrichioides* and *F. poea* in millet have been associated with the human disease alimentary toxic aleukia that was first reported in Russia in the 19th century (Ueno 1983). Cold, moist grain storage appears to favour growth of these fungi.

Fusarium with the potential to produce Type A trichothecenes such as *F. equiseti*, *F. semitectum*, *F. acuminatum* have been isolated from maize and maize soils in Australia (Wearing and Burgess 1978; Watson *et al*. 2006) but their relatively low prevalence in maize suggests that the risk of contamination with type A trichothecenes is also low and probably confined to cooler and wetter districts. Poisoning of livestock by type A trichothecenes has not been diagnosed in Australia and problems with livestock production (vomiting and reduced feed intake, particularly by pigs) are also uncommon (Moore *et al*. 1985; Tobin 1988). On the other hand, type B trichothecenes tend to be produced by *Fusarium* spp., such as *F. graminearum* and *F. culmorum*, that favour warm to temperate climates that occur in parts of Australia.

F. graminearum has a wide distribution in Australian grain crops in warmer regions, causing stalk and ear rots of maize, while a closely related fungus *F. pseudograminearum* causes crown rot of wheat, barley and triticale, but rarely affects the grain (Francis and Burgess 1977). *F. culmorum* is more common on wheat than maize and prefers cooler latitudes in Australia (Burgess *et al*. 1981). There are two different 'chemotypes' of *F. graminearum*; those producing predominantly DON and its acetylated derivatives, and those that produce predominantly NIV and its acetylated derivatives (Ichinoe *et al*. 1983). Both chemotypes occur in wheat, barley and sorghum in southern Queensland and northern New South Wales but DON producers predominate, whereas only NIV producers have been found on maize in Far North Queensland where wheat is not grown (Blaney and Dodman 1988, 2002). Both chemotypes also produce ZEA. *F. pseudograminearum* and *F. culmorum* also produce DON and ZEA.

Infection of maize by *F. graminearum* is favoured by warm, wet conditions during flowering and persistently wet weather during maturation, which are common in Australia only in wetter localities on the tablelands of Far North Queensland and the north coast of New South Wales. Infection of wheat, barley and triticale is also associated with warm, wet conditions during flowering, but particularly in crop rotation with maize and perhaps sorghum. Except in the small localities mentioned, these conditions are also unusual in Australia and infections are associated with unusually wet springs combined with transient increases in maize cropping.

As with ZEA, monitoring of pink-purple discoloured kernels in grain is a useful screening test. In samples collected in the worst affected regions of the northern tablelands in Queensland in 1983 and 1984, the average concentration of NIV in samples with <0.25% purple kernels was 0.13 mg/kg, in samples with 0.25–1% purple kernels 0.66 mg NIV/kg, and in samples with >1% purple kernels 1.21 mg NIV/kg with the maximum of 2.5 mg NIV/kg detected in a sample with >2% of purple kernels (B. J. Blaney, unpubl. data). Continued breeding of maize hybrids for resistance to ear rot in the 20 years since those surveys has probably reduced the extent of contamination.

In wheat, barley and triticale, head blight occurs at quite low prevalence in wetter regions where maize (and sorghum) is also grown. Blaney *et al*. (1987) reported results of a survey of wheat grown in south-east Queensland in 1983–85 including all regions where head blight is significant and in a year where rainfall had been unusually high. Sixty-two of 1291 wheat delivery samples showed physical evidence of head blight (bleached, shrunken grain, some with dark pink tips). Samples pooled according to receipt depot and grade had low concentrations of DON (<0.12 mg/kg) with the exception of one pooled sample that contained 1.7 mg/kg. The Australian Wheat Board has conducted surveys since 1995, which confirm the overall pattern of low prevalence of DON in small grain with detections predominantly confined to certain localities in seasons with unusually wet springs. Human exposure to the type B trichothecenes DON and NIV from wheat is not significant in Australia (Tobin 1988; Webley and Jackson 1998).

In the USA 1 mg/kg DM is the most stringent advisory level for DON in finished wheat products (USFDA 2010). Grain and grain by-products for ruminating cattle and chickens are allowed 10 mg DON/kg DM provided they supply no more than 50% of the total diet (5 mg/kg DM in the total diet). Grain and by-products for pigs are allowed 5 mg DON/kg DM provided they supply no more than 20% of the total diet (1 mg/kg DM in total diet). For all other animals, grain and by-products can contain 5 mg DON/kg DM but not exceed 40% of the total diet (2.5 mg/kg DM in the total diet) (Park and Troxell 2002).

There are no current regulations or standards applicable for DON or NIV in Australian grain except for tolerances of 'nil' pink grains in industry grain receival standards. The GTA standard for all maize grades currently contains a 'nil' tolerance for Fusarium (pink) fungal-stained grain (GTA 2011). Regular testing of maize for DON and NIV does not appear to be warranted except in maize grown in areas of known risk, and checking for pink/purple grain in the first instance will further reduce the risks.

The potential for residues in animal products has been reviewed (EFSA 2004*f*; MacLachlan 2011). DON is well absorbed and metabolised into less toxic products. Elimination occurs by renal and biliary excretion and only trace amounts are transferred into tissues, milk and eggs.

Several studies in North America have indicated that feed naturally contaminated with DON has more impact than when pure DON is administered to animals (Forsyth *et al*. 1977; Foster *et al*. 1986; Rotter *et al*. 1994; Trenholm *et al*. 1994). In pigs a temporary reduction in feed intake was observed at 0.35 mg DON/kg feed but no lasting effect has been shown at 0.6–0.9 mg DON/kg in feed (Friend *et al*. 1982; Young *et al*. 1983; Bergsjø *et al*. 1993; Øvernes *et al*. 1997). The tolerance to NIV was not reviewed by EFSA (2004*f*) although it was acknowledged that feeds containing DON could also contain NIV and acetyl derivatives of DON and NIV.

The tolerance of pigs to both DON (Williams *et al*. 1988) and NIV (Williams and Blaney 1994) in naturally contaminated grain in Australia has been tested. Results were similar in type and magnitude to those reported elsewhere. Vomiting at high intakes (DON only) and persistent feed refusal (with both DON and NIV) were the only adverse effects noted with the tolerance ~1 mg/kg; feed conversion was only affected when levels exceeded 8–9 mg DON/kg feed. It is considered that a guidance level of 0.5 mg/kg DM has a safety margin and is attainable in Australia pig diets without serious impact on grain and feed producers. It is noted that the slightly reduced feed intakes has more impact on young grower pigs than in older pigs which are often restrictively fed. The USA guideline is 1 mg/kg DM in the total diet of pigs (USFDA 2010).

Chickens have been reported to be more tolerant to DON than pigs with no effects on feed intake and growth until dietary concentrations reach 14 mg/kg although slight effects on liver and gizzard weights of chickens fed 9 mg/kg were reported (EFSA 2004*f*). However, Australian broilers have shown a little more susceptibility. Using naturally contaminated Australian wheat, chickens offered a choice between diets containing 12 mg DON/kg and control diets strongly selected against the DON-containing diet and when given no choice, intakes were down and daily gain was reduced by 12% (Mannion and Blaney 1988). Daily gain also declined by 3–8% in chickens fed maize-based diets containing 3–6 mg NIV/kg (Kopinski *et al*. 1991). A guidance level of 2 mg/kg DM, rather than the United States Food and Drug Administration guideline of 5 mg/kg DM in respect to either DON or NIV in the total diet of broilers allows a safety margin. Ducks and hens also appear to tolerate 3–5 mg DON/kg (EFSA 2004*f*) and a guidance level of 2 mg/kg DM is suitable for these species.

Cattle are also considered fairly tolerant to DON. Studies in Canada have shown 1.5 mg/kg was tolerated but transient reductions in feed intake were noted with 6.4 mg/kg (Trenholm *et al*. 1984). In Australia, severe feed refusal, depression and scouring was observed in calves fed triticale subsequently found to contain 30 mg DON/kg (B. J. Blaney, unpubl. data). A guidance level of 2 mg/kg DM in respect to either DON or NIV in the total diet appears suitable compared with the USA guideline of 10 mg/kg DM, with 5 mg/kg DM for young animals (USFDA 2010). Sheep tolerated 4–5 mg/kg without significant effects (EFSA 2004*f*). The USA guideline of 2 mg/kg DM is considered applicable to sheep and species not mentioned above. Guidance levels for the sum of DON, NIV and their acetyl derivatives in the total diet are listed in Table 7.

Ergot alkaloids

Ergot alkaloids are a diverse group of up to 40 compounds, comprising ergopeptides, clavines, lysergic acids and lysergic acid amides, produced by members of the fungal family Clavicipitaceae. Members of the family noteworthy on the basis of toxicity to animals include *C. purpurea* (infecting rye, rye grass, wheat, barley and oats), *C. paspali* (infecting *Paspalum* spp. of grasses), *C. fusiformis* (infecting millet) and *C. africana* (infecting sorghum). Other toxic members of the Clavicipitaceae include endophytic fungi of grasses, such as *Neotyphodium coenophialum*-infecting tall fescue and *N. lolii*-infecting perennial rye grass.

Ergot alkaloids are produced as the fungus develops, but are eventually concentrated in the hard-bodied resting stage of the fungus, the sclerotium. These sclerotia can fall to the ground and repeat the life cycle, or be harvested with the grain. Mature sclerotia vary in number and size from a few millimetres to more than 4 cm long according to the host plant (Kamphues and Drochner 1991; Meyer 1999) and differ in mass from a few grams to 25 g for 100 sclerotia. Ergot sclerotia also vary in colour from white (*C. tripsaci*), to brown (*C. glabra*) to yellow (*C. hirtella*) and purplish-brown (*C. purpurea*). Sclerotia also show significant differences in their total alkaloid content, which varies between 0.01 and 0.21% (Lorenz 1979; Wolff 1989) and in the alkaloid profile.

Rye ergot: (*C. purpurea*) can produce a range of alkaloids, including ergotamine, ergosine, ergocristine, ergocryptine, ergocornine and ergonovine (ergometrine) with the composition and content of sclerotia from different countries varying considerably (Young and Chen 1982). The alkaloid profiles of ergot sclerotia from south-western and eastern Australia are very similar. Ergotamine and ergocryptine are the major components with lesser concentrations of ergocornine and ergosine (Blaney *et al*. 2009). Ergot alkaloids may be converted to their C8 epimers on storage (i.e. epimerisation at position C8). A large proportion of the alkaloid content of sclerotia is represented by these epimers and it is unclear how much is present in developing sclerotia and how much is a result of isomerisation during storage. While relatively inactive in laboratory animal models (Stoll 1952; Goodman *et al*. 2011) there is insufficient information on the potential for the epimers to be converted back to active isomers in the rumen and risk assessment should consider the total alkaloid content of feed (Blaney *et al*. 2009).

Rye ergot infects annual rye grass (*Lolium rigidum*), particularly in south-eastern and south-western Australia (Reed *et al*. 2005). Infection of cereal crops, including rye, oats, wheat and barley is rare in Australia mainly due to the dry conditions prevailing during flowering of these crops but occurs in Europe and North America (Blaney *et al*. 2009). Consequently poisoning of livestock occurs only occasionally and in a few localities either as a result of grazing infected rye grass or if rye grass is not controlled in wheat or barley crops and grain becomes contaminated with ergot sclerotia during harvest (Blaney *et al*. 2009). Management of contamination of stock food might need to consider levels in grain and in hays prepared from infected pastures. Only the situation of ergot bodies in grain is considered here.

There is negligible transfer of rye ergot alkaloids from feed to edible tissues, milk or eggs (Whittemore *et al*. 1976, 1977; Parkheava 1979; Young and Marquardt 1982; Wolff *et al*. 1995; Mainka *et al*. 2005; Schumann *et al*. 2007, 2009). There is insufficient risk to human health in Australia from ergot alkaloids and their metabolites in food of animal origin to justify regulation of ergot sclerotia in animal feed on the basis of human health risks.

Ergot alkaloids affect animal production and setting of guidance levels for ergot and ergot alkaloids in stock foods is justified on this basis. Ruminants appear to be more sensitive to the effects of ergot than monogastric animals. The threshold tolerance of cattle and sheep for ergot alkaloids is not clear. In the few Australian cases of livestock poisoning severe hyperthermia was observed in ruminants fed 1–3 mg alkaloids/kg. To avoid severe poisoning it was suggested (Blaney *et al*. 2009) that the total alkaloid content of feed should be restricted to <0.4 mg/kg, which for sclerotia with an alkaloid content of ~0.2% equated to the existing 0.02% limit for ergot sclerotes in stock food in Queensland (Qld 1997).

In a study in Australia, Bourke (2003) produced hyperthermia in cattle fed at the estimated equivalent of 1 mg alkaloids/kg in feed. However, he concluded that stock exposure to sunlight appears to be a critical factor in a particularly lethal form of hyperthermia in cattle and sheep and that toxins other than ergot alkaloids could be involved such as the ergochromes (Franck 1969; Buchta and Cvak 1999). Bourke (2003) suggested that feed

likely to contain rye ergot should be avoided for ruminant feed, particularly in feedlot rations. For practical purposes it is preferred to propose a low level rather than a zero tolerance and a level of 0.01% ergot sclerotia in the total diet of cattle, goats and sheep is proposed, with an equivalent alkaloid content limit of 0.2 mg/kg. This limit is lower than that suggested by Blaney *et al*. (2009), and lower than current state regulations, but given the very low frequency of contamination of bulk grain in Australia, it appears achievable by industry and consistent with the principle of as-low-as-reasonably-achievable.

Pigs and poultry are a better option for use of lightly contaminated grain than ruminants apart from the serious risk of agalactia in sows fed ergot before farrowing, due to the inhibitory effects of ergot alkaloids on release of prolactin (Anderson and Werdin 1977; Kopinski *et al*. 2007). In one Canadian study (Dignean *et al*. 1986) milk production was not affected when sows were fed 0.2% rye ergot (4.5 mg alkaloids/kg of diet) from breeding until weaning, but the authors noted that their results were apparently at variance with other studies showing agalactia produced by lower ergot concentrations. Studies with sorghum ergot fed to sows before farrowing, found adverse effects on milk production at alkaloid concentrations of 1.4–7 mg/kg (Kopinski *et al*. 2007), but higher concentrations were tolerated after lactation had commenced (Kopinski *et al*. 2008*c*). Nevertheless a concentration of 0.02% rye ergot (0.4 mg alkaloids/kg) should provide an adequate safety margin for weaner and breeder pigs. The tolerance of non-lactating pigs is also not clear but a Canadian study (Oresanya *et al*. 2003) suggested maximum tolerances of 0.1% ergot (2 mg alkaloid/kg) and 0.05% based on growth rates and feed intakes respectively for weaner pigs (7–20 kg liveweight). Other studies have shown that grower and finisher pigs are more resistant and 10–15 mg/kg can be tolerated with only minor effects on feed intakes that can be masked with palatable ingredients (Whittemore *et al*. 1977; Mainka *et al*. 2005; Kopinski *et al*. 2008*a*, 2008*b*, 2008*c*). One Australian study (Bakau *et al*. 1988) found reductions in growth rate and feed intake of pigs fed 0.75% ergot (alkaloid content not reported), which were exacerbated by higher temperatures (35°C). On the basis of the above a level of 0.1% rye ergot (2 mg alkaloids/kg) is proposed for grower and finisher pigs.

Various studies have shown that chickens are tolerant of rye ergot at a concentration of 0.5%, but 1–5% progressively affects feed intake, feed conversion and growth rates (Rotter *et al*. 1985*a*, 1985*b*; Bakau and Bryden 1987). A guidance level for poultry of 0.2% rye ergot (4 mg alkaloids/kg) provides an additional safety margin. Guidance levels for rye ergot or total rye ergot alkaloids in the total diet are listed in Table 7.

Sorghum ergot: *C. africana* is one of three species of ergot-infecting sorghum and related species, the others being *C. sorghi* and *C. sorghicola*, but only *C. africana* has so far been identified in Australia (Blaney *et al*. 2006). The main alkaloid produced by *C. africana* is dihydroergosine (DHES) (>80%), with dihydroelymoclavine and festuclavine as minor components (Blaney *et al*. 2003) and the three alkaloids occur in fairly constant relative proportions. As with other ergot fungi, sorghum ergot infects sorghum plants during flowering, and begins to replace the ovaries of infected flowers with a fungal body, the sphacelium. About 1 week after flowering, the sphacelium forces the floret open and infection is signalled by copious release of sticky honeydew (Frederickson *et al*. 1993). Gradually, in parallel with grain development which occurs over the next 4 weeks, this sphacelial tissue is replaced by harder sclerotial tissue (Frederickson and Odvody 1999). Only occasionally, in circumstances not clearly understood, a fully mature sclerotium may be formed (the hard walled, resting stage of the fungus). Low concentrations of DHES are produced by the sphacelial tissue and are present in honeydew (1–10 mg/kg, Blaney *et al*. 2006), but by far the most is produced by the sclerotial tissue (Mantle 1973; Blaney *et al*. 2006). Following its initial discovery in Australia in 1996 (Ryley *et al*. 1996), sorghum ergot was found to be present in all significant sorghum-growing areas of Australia with infection mainly associated with late-planted crops flowering in cold weather when pollination is impaired. Sorghum ergot contamination of grain differs from that of rye ergot in that fully mature sclerotia are rarely formed, and 'ergot' in sorghum is defined as the total weight of all ergot bodies, including mature sclerotia, sphacelia with glumes attached, and sphacelia overgrown with sporodochia of the ergot saprophyte *Cerebella* spp. The average concentration of alkaloids in 'sorghum ergot' are therefore much less (0.02% compared with 0.2% in mature sclerotia), but the relationship between 'ergot' and alkaloid concentration is poor (Kopinski *et al*. 2008*b*). In consequence, % ergot only provides a guide to contamination, which should be supported with alkaloid assay.

No residue data are available in milk or meat, but studies with related compounds (rye ergot alkaloids) show rapid metabolism in the rumen to base compounds (lysergic acid, etc), which retain pharmacological activity. One study has shown that no residues of DHES (<5 μg/kg) occur in eggs from hens fed high concentrations of sorghum ergot (Dingle *et al*. 2003). Consequently, there appears no reason to restrict sorghum ergot alkaloids in livestock feed in order to protect human health, unless concern should be raised in the future over the fate of alkaloid metabolites. Restrictions are therefore only warranted to protect livestock health.

Sorghum ergot alkaloids have similar effects as rye ergot alkaloids in reducing circulating prolactin concentrations, with severe impact on milk production of sows (Kopinski *et al*. 2007, 2008*c*) and cows (Moss *et al*. 1999; Blaney *et al*. 2000*c*). They also have vasopressor effects, reducing peripheral blood circulation and interfering with heat regulation in cattle (Blaney *et al*. 2001).

The growth and fattening of steers managed under feedlot conditions were severely affected at sorghum-ergot alkaloid concentrations of 1.1 mg/kg and over (Blaney *et al*. 2011). In experiments conducted by McLennan *et al*. (2001) in summer–autumn and in winter–spring, Hereford steers were fed a grain-based concentrate ration with alkaloid concentrations in the grain component of 1.5–12 mg DHES/kg. Ergot increased susceptibility of the steers to heat stress (shown by excessive salivation, panting, and excessive drinking/urination). Even very low concentrations of ergot alkaloids impair performance of cattle in feedlots, and the effects are more severe during hot, humid weather. It is proposed that sorghum ergot alkaloids in the total diet of non-lactating ruminants should be restricted to 0.3 mg alkaloids/kg. This will most commonly be equivalent to 0.1% ergot, but could rarely but conceivably be present in a sample

containing 0.02% of mature sclerotia (Kopinski *et al*. 2008*c*). Consequently, the proposed guidance limit for ergot should be supported by physical observation of the presence or absence of mature sclerotia, and by alkaloid assay when risks are higher.

Dairy cows are also very susceptible to sorghum ergot, with hyperthermia, reduced feed intakes and pronounced decline in milk production (Moss *et al*. 1999; Blaney *et al*. 2000*a*). In cows exposed to sorghum ergot, intakes of 2 and 4 mg/head.day produced little effect on grain consumption and milk production while at 8 and 16 mg/head.day milk yields declined from the start of feeding. Sorghum intakes were 5 kg/head.day, putting the tolerated level at 0.8 mg/kg. Assuming the total feed intake including pasture to be ~15 kg DM/day, the tolerated level in the total diet would be 0.3 mg alkaloids/kg. High temperatures in subtropical Australia are already a major constraint on dairy cow production, and ergot alkaloids can exacerbate this. To allow a safety margin to account for the effects of temperature, a guidance maximum level of 0.03% ergot (0.1 mg alkaloids/kg) is proposed.

When fed to pre-farrowing sows, 1.5% ergot (7 mg alkaloids/kg) caused complete agalactia, while minor reductions in milk production were observed at concentrations down to 0.3% ergot (1.4 mg alkaloids/kg) (Kopinski *et al*. 2007). After lactation was in full flow sows were less susceptible, but 3% ergot (16 mg alkaloids/kg) still reduced milk production (Kopinski *et al*. 2008*c*). When diets containing 0.3% sorghum ergot (1.3 mg alkaloid/kg) were fed to sows from 16 weeks before farrowing until weaning of piglets 4 weeks post-farrowing, no adverse effects were observed, although a trend for reduced plasma prolactin in first litter gilts suggested that this was near the level of tolerance (Kopinski *et al*. 2008*c*). The suggested guidance maximum level of 0.1% ergot or 0.3 mg alkaloids/kg provides a safety margin.

Grower and finisher pigs were shown tolerate up to 35 mg/kg alkaloids in short-term feeding (Blaney *et al*. 2000*b*) and 10 mg alkaloids/kg (1–4% ergot, depending on batch) over the entire grower-finisher period (Kopinski *et al*. 2008*a*, 2008*b*), providing that feeds are formulated to minimise nutritional deficiencies and some potential decrease in palatability. A guidance maximum level of 1% ergot (3 mg alkaloids/kg) provides a safety margin. Young weaner pigs appear to be more susceptible to the effects of ergot alkaloid than older pigs, and lower limits for weaner diets are suggested.

Broiler chickens have been shown to be fairly resistant to the effects of sorghum ergot (Blaney *et al*. 2001). At alkaloid concentrations ~15–20 mg/kg there are no significant effects on growth, feed conversion or mortality (Bailey *et al*. 1999). At 30 mg/kg, there can be some depression in feed intake and growth, accompanied by slight increases in heart and liver weight. Layer hens also can tolerate sorghum ergot. Over a 6-week period where hens were fed 0, 6, 12 or 24 mg alkaloids/kg there were significant decreases in egg production and egg mass at the 24 mg/kg level, but not at lower concentrations. It was concluded that 6 mg alkaloid/kg would not significantly affect production (Dingle *et al*. 2003). A guidance maximum level is proposed for poultry at 6 mg alkaloids/kg DM.

A standard exists in Queensland for ergot (*Claviceps* spp.) other than sorghum ergot (*C. africana*) of 200 mg/kg (0.02%) and for sorghum ergot (*C. africana*) of 3000 mg/kg (0.3%) (Qld 1997).

The currently legislated stock feed limit of 0.3% ergot by weight in sorghum grain equates to ~1 ergot body per 100 seeds or 30 ergot bodies per 100 g grain. The GTA standard is 0.1% sclerotia by weight for stock feed intended for feedlot cattle with a limit of 0.3% for all other uses (GTA 2011). Deliveries of sorghum with sclerotia concentrations higher than 0.3% will be rejected by grain merchants and those higher than 0.1% will be rejected by cattle feedlotters. Most commonly, a sorghum sample containing 0.3% ergot bodies will contain ~1 mg alkaloid/kg (Kopinski *et al*. 2008*b*).

If ergot-contaminated grain is milled, between 80 and 90% of the ergot can be directed to the bran and shorts feed streams. These fractions are regularly destined for animal feed but there are no available data on contamination levels. Care needs to be taken that grain ‘dockage’ does not ultimately get routed into animal feed.

Phomopsins

Lupinosis is a mycotoxicosis caused by the ingestion of toxins, phomopsins, produced by the fungus *Diaporthe toxica*, which colonises lupin plants (Allen 1987; Williamson *et al*. 1994). Western Australia is the major lupin producer in the world, accounting for 70% of world production and lupin fodder and stubble has been used as a feed source in this region, primarily for sheep (Allen 2009). The main source of exposure of livestock has been through grazing on or feeding of fodder, with few documented reports of lupinosis from feeding seed, as commercial grading of seed to remove discoloured seeds (which are toxin containing) effectively manages this source (Petterson *et al*. 1997). The introduction of phomopsis-resistant lupins has also greatly reduced the risk of lupinosis such that lupinosis is no longer considered a disease of major importance to livestock producers in Western Australia (Allen 2009). There are insufficient data available at this stage to propose guidance levels for phomopsins.

Bacterial toxins

Another group of bacterial toxins present in some pasture hay that is of importance to the livestock industry is corynetoxins.

Corynetoxins

Corynetoxins are a family of nucleosidyl glycolipid antibiotics that are produced by the bacterium *Rathayibacter toxicus* that can colonise the seed heads of grasses. A nematode vector (*Anguina funestra*) forms a nematode gall in the seed head of the grass and in this way carries the bacteria to the plant. Corynetoxins are produced as the plant senesces towards the end of the growing season. Ingestion of corynetoxin-contaminated feed then produces the neurological diseases annual ryegrass toxicity and flood plain staggers (Bryden *et al*. 1994; Edgar *et al*. 1994).

Corynetoxins are best managed on farm through the control of rye grass in pastures with herbicides, biological control measures such as the twist fungus (*Dilophospora alopecuri*), which can reduce the prevalence of the nematode vector and the use of resistant varieties of rye grass. Currently available data are not

sufficient to allow a guidance maximum level to be proposed for corynetoxins.

Interpretation of guidance maximum levels

Analytical methods and sampling

In utilising maximum guidance levels care needs to be taken to ensure any analytical results relied upon to determine the suitability of feed are sufficiently accurate. Whitaker (2003) has reviewed issues of particular relevance to analysis of feed samples. Contamination of feed is often not uniform. This means that obtaining a representative sample of the load is critical in getting an accurate estimation of the extent of contamination. Additionally, a variety of test methods may be available for a particular contaminant, including enzyme-linked immunosorbent assay and liquid chromatography. Each test varies in accuracy, specificity and variability as well as speed of analysis, complexity and cost. Test results will vary when an analysis is conducted multiple times, and results will exhibit further variation when conducted by different analysts in different laboratories. Laboratory reports should indicate the uncertainty inherent in the final reported value. It is very important to ascertain if the method used by a particular laboratory will be sufficiently accurate. The uncertainty about results must be factored into risk management decisions. In Australia, the National Association of Testing Laboratories (NATA) accredits laboratories to perform specific tests in compliance with Australian Standard AS ISO/IEC 17025:2005. In order to be accredited by NATA, laboratories must validate their test methods to objectively demonstrate that the results produced by those methods are fit for purpose. Accredited laboratories must also estimate, and if requested, report the measurement uncertainty associated with their test results. It must be recognised that the uncertainty in results reported by laboratories only takes into account the potential variability in the laboratory analysis and does not include variation attributable to sampling.

Application of guidance levels to field situations

The use of recommended maximum guidance levels is illustrated below for lead. A feed or feed ingredient sampled for lead is considered acceptable if the concentration found is at a level below the relevant guidance maximum level. Where a guidance level has not been proposed for a feed ingredient the calculation is based on the total diet and includes feed as well as exposure from ingested soil. The examples below illustrate the use of relative bioavailability factors, however in most cases these are not available and would be assumed to be 100% (i.e. $r = 1$).

In the following hypothetical example a feed premix for pigs has been analysed and found to contain 43 mg lead/kg DM. The inclusion rate of the premix in the complete feed is 5%. There are no other sources of exposure as the pigs are not fed forage and have no access to soil. The form of lead in the premix is known to be lead oxide with the relative bioavailability assumed to be the highest listed in the Supplementary Material for lead oxide (75%). The concentration of lead in the total diet is then $43 \times 0.05 \times 0.75 = 1.61$ mg lead/kg DM. Concentrations of lead in tissues at slaughter should be less than relevant standards as the concentration calculated for lead in the total diet is below the guidance maximum level (of 5 mg/kg).

Calculations for ruminants differ in that they usually have access to forage as well as soil. In the following calculation a premix containing 43 mg lead/kg DM is incorporated in the complete feed for cattle at 5%. It is assumed the cattle total diet comprises 50% complete feed, 45% forage containing 0.1 mg lead/kg DM and 5% soil containing 1 mg lead/kg soil. The relative bioavailabilities are 75% for lead present as lead oxide in the premix, 100% for forage and 25% for soil. The concentration of lead in the total diet is then $0.5 \times (43 \times 0.05 \times 0.75) + 0.45 \times (0.1 \times 1) + 0.05 \times (1 \times 0.25) = 0.86$ mg lead/kg DM, below the guidance maximum level, suggesting the presence of lead in the premix does not represent a concern.

If feeds that exceed the maximum guidance levels listed here are fed to livestock, the livestock may need to be managed to ensure concentrations of contaminants are below relevant standards at slaughter, for example by ensuring a period where livestock are not exposed to the contaminant to allow tissue concentrations to decline to acceptable levels before slaughter.

Conclusion

A modern food production chain requires all participants to be confident in suppliers of inputs. In this regard transfer of information between participants is important to enable responsible decisions to be made in order to effectively manage risks.

The FeedSafe program (http://www.sfmca.com.au/feedsafe/about_feedsafe/, accessed 15 October 2012) followed by most commercial feed mills in Australia has a quality assurance program that is audited by third party auditors on an annual basis. FeedSafe requires a documented raw material sourcing and purchasing program to be implemented to minimise potential product quality and safety risks.

The suggested guidance maximum levels for various contaminants in livestock feed should serve as a useful resource for those involved in feed production and assist in the development of meaningful Hazard Analysis Critical Control Points programs for control of feed contaminants.

There are several options for the management of contaminants in the production of food of animal origin. For example, purchase specifications can be developed for feed and feed ingredients, vendor declarations can be obtained from feed suppliers, restrictions can be put in place to manage the level of inclusion of a feed item in the total ration or diet, or feeding can be restricted to certain classes of animals based on the concentration of the contaminant present. Finally, animals can be placed on ‘clean feed’ for a period before slaughter.

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